Cerebellar circuitry and auditory verbal hallucinations

Neuroscience and Biobehavioral Reviews 118 (2020) 485–503

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Review article

Cerebellar circuitry and auditory verbal hallucinations: An integrative synthesis and perspective

Ana P. Pinheiro[a,b,*], Michael Schwartze[b], Sonja A. Kotz[b,c]

[a] Faculdade de Psicologia, Universidade de Lisboa, Lisboa, Portugal
[b] Faculty of Psychology and Neuroscience, Department of Neuropsychology & Psychopharmacology, Maastricht University, Maastricht, The Netherlands
[c] Department of Neuropsychology, Max Planck Institute for Human Cognitive and Brain Sciences, Leipzig, Germany

ARTICLE INFO

Keywords:
Auditory verbal hallucinations
Forward model
Cerebellum
Cognition
Psychosis continuum

ABSTRACT

Auditory verbal hallucinations (AVH) — experienced as voice hearing independent of a corresponding external sound source — are a cardinal symptom of psychosis. Approximately 6-13% of healthy individuals also experience voice hearing. Despite numerous attempts to explain the neurofunctional mechanisms underlying AVH, they remain notoriously unexplained. However, evidence relates AVH to mechanistic changes in the forward model. This review synthesizes behavioral and neuroimaging studies exploring the central role of cerebellar circuitry in the forward model, with a particular focus on non-verbal and verbal auditory feedback. It confirms that erratic prediction of sensory consequences in voice and sound production is linked to impaired cerebellar function, which initiates AVH and affects higher-level cognitive functions. We propose new research directions linking the forward model to voice and sound feedback processing. We consider this review as a starting point for mapping mechanisms of the forward model to neurocognitive mechanisms underlying AVH.

1. Public significance statement

This systematic review suggests that mechanistic changes in the forward model, linked to impaired cerebellar function, likely contribute to auditory verbal hallucinations in psychotic and nonclinical voice hearers. By bridging behavioral and neural evidence, this review proposes an answer to the persisting question why some people hear voices when there are none and recommends future research directions.

2. Neural bases of voice specificity

Voices are socially the most relevant stimulus in the auditory landscape of humans. They carry speech and nonverbal cues such as dominance or the emotional state of a speaker (Banse and Scherer, 1996; Liu et al., 2012; Paulmann et al., 2013; Pell et al., 2009; Pinheiro et al., 2015). Neuroimaging research further revealed specialized voice patches in temporal cortices located bilaterally along the mid and anterior parts of the superior temporal gyrus/sulcus (Belin et al., 2004). These areas not only respond to actual acoustic input but also to imagined voices (Yao et al., 2012, 2011) and to experiencing auditory verbal hallucinations [AVH] (Barkus et al., 2007; Horga et al., 2014b; Linden et al., 2011; Sommer et al., 2008).

Why do some people hear voices in the absence of actual acoustic input? Approximately 70 % of persons diagnosed with psychosis experience AVH, which makes verbal hallucinations more common than visual hallucinations (Bauer et al., 2011). However, AVH also feature in other psychiatric disorders such as bipolar disorder or major depressive disorder (Toh et al., 2015), as well as in 6–13 % of the healthy general population (Beavan et al., 2011; Linscott and van Os, 2013; Waters and Fernyhough, 2017). AVH in psychotic and nonpsychotic individuals seem comparable as they engage similar cognitive mechanisms and brain regions (e.g., Daalman et al., 2011; Sommer et al., 2010). In line with this observation, the *psychosis continuum hypothesis* postulates that nonclinical individuals can display psychotic-like symptoms (e.g., Verdoux and Van Os, 2002). This perspective acknowledges dynamic transitions from normal to aberrant perception leading to psychosis (Van Os et al., 2009).

AVH are usually experienced as indistinguishable from real voices with specific acoustic properties, including volume, pitch, spatial location, gender, age, or accent structure (Larøi et al., 2012; McCarthy-Jones et al., 2014b; Nayani and David, 1996). They typically relate to agents with a specific identity (e.g., “the voice of God”), refer to the voice hearers and address them in the second grammatical person (Larøi et al., 2012; McCarthy-Jones et al., 2014b; Nayani and David, 1996). It is not uncommon that AVH subsume more than one voice conversing with each other (Larøi et al., 2012; McCarthy-Jones et al.,

* Corresponding author at: Faculdade de Psicologia, Universidade de Lisboa, Alameda da Universidade, 1649-013 Lisboa, Portugal.
E-mail address: appinheiro@psicologia.ulisboa.pt (A.P. Pinheiro).

https://doi.org/10.1016/j.neubiorev.2020.08.004
Received 7 February 2020; Received in revised form 30 June 2020; Accepted 7 August 2020
Available online 15 August 2020

2014b; Nayani and David, 1996). AVH often have an emotional quality, but their content seems to differ in psychotic and nonclinical voice hearers: hallucinated voices in psychosis are frequently perceived as harsh, authoritative, or aggressive, and often convey negative and malevolent content (e.g., Copolov et al., 2004); the content of voices tends to be less negative in nonclinical voice hearers (Baumeister et al., 2017). They are perceived as similar to hearing other people speak, varying in length from single words to complex conversations and may appear as dominant or powerful (Larøi et al., 2012; Nayani and David, 1996; Waters and Fernyhough, 2017). Due to their acoustic, linguistic, and social properties, voice hearers may establish a relationship with these voices (Beavan, 2011; Bell, 2013; Birchwood et al., 2000; Sorrell et al., 2010; Vaughan and Fowler, 2004).

Despite many efforts to explain this puzzling phenomenon, the neurocognitive mechanisms underlying AVH remain largely elusive. Different models have been proposed, focusing on different mechanisms to account for AVH and that include impaired intentional inhibition of traumatic memories (e.g., Morrison and Baker, 2000; Waters et al., 2006), altered auditory imagery (e.g., Aleman et al., 2002), or faulty self-monitoring or source identification of internal events (Bentall, 1990; Brébion et al., 2000, 1996; Frith and Done, 1988) such as inner speech (Allen et al., 2007a; Feinberg, 1978; Fernyhough, 2004; Johns et al., 2010, 2001; McGuire et al., 1995). The diversity of the proposed mechanisms reflects the complexity of the phenomenon of hearing voices, also documented by symptom-capture functional magnetic resonance imaging (fMRI) studies that compare periods of presence and absence of AVH. These studies reported consistent alterations in a distributed network of fronto-temporal cortical and subcortical brain regions, including those involved in speech generation and perception, conscious recollection of episodic events or self-monitoring: superior temporal gyrus (Jardri et al., 2011; Lennox et al., 2000; Shergill et al., 2000), middle temporal gyrus (Hoffman et al., 2008; Shergill et al., 2000), inferior frontal gyrus (Raij et al., 2009; Shergill et al., 2000), middle frontal gyrus (Lennox et al., 2000), anterior insula (Hoffman et al., 2008), anterior cingulate (Hoffman et al., 2008; Shergill et al., 2000), basal ganglia (Silbersweig et al., 1995), and hippocampal formation (hippocampus and parahippocampal cortex; Diederen et al., 2010; Hoffman et al., 2008; Shergill et al., 2000; Silbersweig et al., 1995). Similar brain regions were activated during AVH in nonclinical participants (Diederen et al., 2012; Linden et al., 2011). Resting-state functional connectivity analyses identified patterns of aberrant activity in circuits involving similar brain regions: superior and middle temporal gyrus (Clos et al., 2014; Diederen et al., 2013; Hoffman et al., 2011; Sommer et al., 2012; Van Lutterveld et al., 2014; Wolf et al., 2011), inferior frontal gyrus (Clos et al., 2014; Diederen et al., 2013; Hoffman et al., 2011; Sommer et al., 2012), anterior cingulate (Shinn et al., 2013; Van Lutterveld et al., 2014; Vercammen et al., 2010; Wolf et al., 2011), anterior insula (Clos et al., 2014; Manoliu et al., 2014), basal ganglia (Hoffman et al., 2011; Sorg et al., 2013), and hippocampal formation (Clos et al., 2014; Diederen et al., 2013; Rotarska-Jagiela et al., 2010; Shinn et al., 2013; Sommer et al., 2012).

Others have proposed that AVH result from mechanistic changes in the *forward model* of the motor system (e.g., Frith et al., 1998), a computational process that partly relies on the cerebellum (Knolle et al., 2013, 2012; Person, 2019; Schlerf et al., 2012; Tanaka et al., 2020; Wolpert et al., 1998). This influential account has received support by an increasing number of studies using behavioral (e.g., Blakemore et al., 2000; Sugimori et al., 2011), electroencephalography (e.g., Heinks-Maldonado et al., 2007; Pinheiro et al., 2018), structural (e.g., Huang et al., 2015; Neckelmann et al., 2006) and fMRI (task-activation: Horga et al., 2014b; Powers et al., 2017; resting-state functional connectivity: Alonso-Solís et al., 2015; Chang et al., 2015; Clos et al., 2014; Mallikarjun et al., 2018; Zhao et al., 2018) methods. Contrary to an inverse model that provides the motor commands that generated a sensory consequence, a forward model predicts the sensory consequences of an action given the current state and motor command (Ito, 1970; Kawato et al., 1987; Miall and Wolpert, 1996). The cerebellum is thought to play a key role in the predictive computations underpinning the forward model (Bastian, 2006; Morton and Bastian, 2006; Nowak et al., 2004; Tanaka et al., 2020; Tseng et al., 2007). In the context of voice production, perceiving one's own voice while speaking involves sensory feedback monitoring in voice production and the rapid detection of discrepancies between expected and actual feedback. That is, the rapid and accurate prediction of the sensory consequences of a motor action is necessary for fast and stable motor control, and a sense of agency (e.g., Haggard, 2017). Dysfunctional monitoring and detection of discrepancies may give rise to misperceptions of the origin of self-generated thoughts and actions (Fletcher and Frith, 2009; Nazimek et al., 2012). Evidence in nonclinical voice hearers has provided further insight on these mechanistic changes (e.g., Pinheiro et al., 2020, 2018), suggesting cerebellar involvement and a shared neurobiological mechanism underlying AVH in psychotic and nonclinical groups. However, many questions remain.

The hypothesis that malfunction of cerebellar circuitry may account for psychotic-like symptoms, including the experience of hearing voices, is not entirely new. The cognitive dysmetria account of psychosis implicates changes in the cerebellum and in its connections with the neocortex (Andreasen et al., 1999, 1998,1996; Andreasen and Pierson, 2008; Schmahmann, 2004; Wiser et al., 1998), which have been documented by clinical, behavioral, and neuroimaging findings (reviewed in Moberget and Ivry, 2019). Accordingly, cerebellar morphology (Moberget et al., 2019) and connectivity (Cao et al., 2018; Cao and Cannon, 2019) were identified as the most important predictors of psychotic symptoms and as the most important differentiator of psychotic patients with *or* without AVH (Chen et al., 2019). Similarly, auditory hallucinations have been reported in the context of focal cerebellar lesions (Bielawski and Bondurant, 2015) or cerebellar neurodegeneration (e.g., spinocerebellar ataxias – Turk et al., 2018; genetic fronto-temporal dementia – Sellami et al., 2018). Notably, cerebellar lesions were found to be associated with hallucinations in the auditory (*vs.* visual) modality (Kim et al., 2019). Notwithstanding, the role of the cerebellum in AVH has remained largely peripheral in the literature so far potentially due to a cortico-centric bias as seen in optimized cortical scanning and analysis protocols that often neglect the cerebellum (Parvizi, 2009). While there is evidence for altered sensory feedback and cerebellar involvement in AVH, links between both types of evidence are scarce. Thereby, the cerebellum's contribution to putative changes within a forward model underpinning AVH remains to be specified.

To address some of these questions, the current review combines behavioral and neuroimaging (electrophysiological and fMRI) evidence on structural and functional characteristics as well as specific changes related to the forward model to further our understanding of AVH. To this end, we synthesize studies exploring the role of the cerebellum in relation to the forward model with studies investigating auditory feedback processing in AVH. These studies typically contrast active sound production (e.g., speaking or button-presses eliciting tones or voices) with passive listening of identical sounds in psychotic or nonclinical voice hearers. We note that there is no monocausal link between the cerebellar function and experiencing AVH but that the cerebellum contributes to this experience as a central node in a network putatively involved in the forward model. The cerebellum is connected to various brain regions, including those involved in speech perception (e.g., superior temporal gyrus), speech generation (e.g., inferior frontal gyrus), and higher-order cognition (e.g., prefrontal cortex) (Brodal, 1978; Schmahmann and Sherman, 1998), which were found to play a critical role in AVH (see also Fig. 1). Therefore, it is critical and timely to look at the contribution of the cerebellum in AVH from a network perspective. We consider this synopsis as a starting point for mapping mechanisms of the forward model to neurofunctional mechanisms underlying the puzzling phenomenon of hearing voices when there are none.

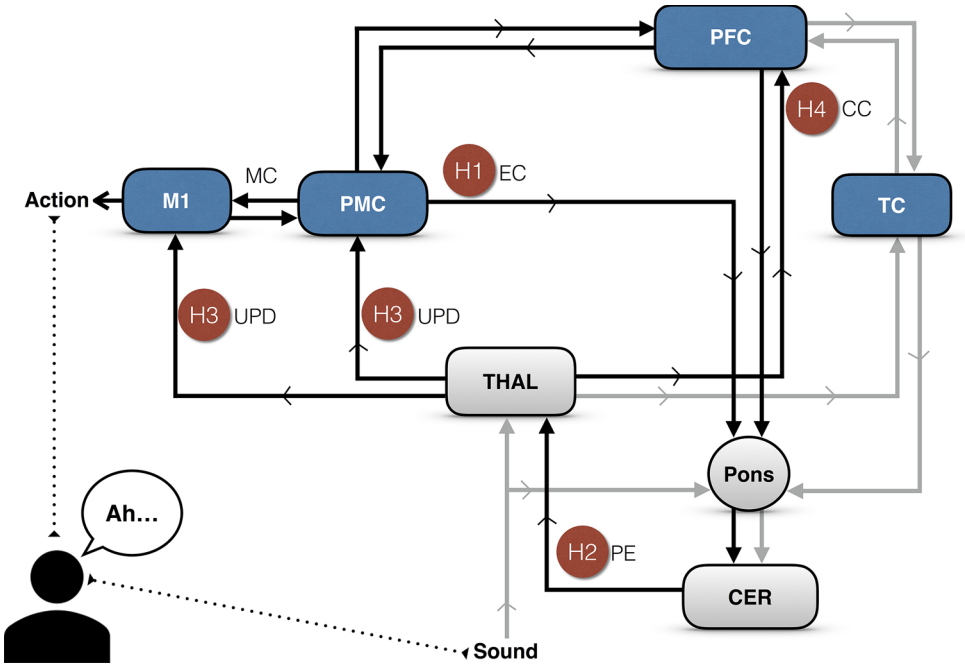

Fig. 1. Circuitry supporting neurofunctional mechanisms underlying action-related sensory feedback in AVH.

Cerebellar-cortical connections include feed-forward loops with obligatory synapses in the pons (cortico-ponto-cerebellar circuits) and feedback loops with obligatory synapses in the thalamus (cerebello-thalamo-cortical circuits) (Ramnani, 2006). Whereas sensorimotor information is relayed to the cerebellar anterior lobe via the caudal pons, cognitively relevant information is relayed to lateral posterior cerebellar regions via the rostral pons (Baumann et al., 2015). One prominent hypothesis is that cortico-cerebellar-cortical loops support not only the control of overt actions, but also the control of internal thought processes and inner speech (Ito, 2008).

When preparing or imagining speaking, activity increases in a number of cortical regions, such as the SMA, IFG, PMC, and M1 (the latter two are shown in Fig. 1). These cortical regions form parallel loops with cerebellar regions. A motor command is ensued by these regions and sent to the peripheral nervous system for action execution. The cerebellum receives the efference copy in parallel to the motor command that is sent to the periphery. Input from the cerebral cortex to the cerebellum plays an essential role in predictive computations thought to underlie the forward model (Tanaka et al., 2020).

When voice feedback is generated, the cerebellum receives auditory input from the temporal cortex (e.g., STS and STG) via cortico-ponto-cerebellar projections (Schmahmann and Pandya, 1991). Importantly, the cerebellum also receives auditory input via its connections to the cerebellum-like dorsal cochlear nucleus (not represented in Fig. 1) during early stages of auditory processing (Huang et al., 1982; McLachlan and Wilson, 2017; Petacchi et al., 2005; Schwartze and Kotz, 2013). The cerebellum (e.g., cerebellar Crus II auditory region) returns connections to the temporal cortex via the thalamus (medial geniculate body) (Pastor et al., 2008). The comparison of expected and actual sensory feedback is thought to take place in the cerebellum (e.g., Miall and Wolpert, 1996); the result of the comparison is used to attribute agency to actions and sensory events (Blakemore and Frith, 2003; Gentsch and Schütz-Bosbach, 2011; Haggard, 2017; Kühn et al., 2011; Synofzik et al., 2010). In the case of a mismatch between expected and actual sensory feedback, error signals may allow the cerebellum to issue a feedback command via the thalamus to premotor areas, primary motor cortex, and selected regions of the prefrontal cortex for sensorimotor control and update of the forward model, as well as to temporal lobe regions. The error signal sent to the temporal cortex results in decreased sensory suppression and, inversely, in increased attention to self-generated feedback. Consequently, attentional demands may increase. Information from the prefrontal cortex also reaches the cerebellum via monosynaptic pathways in the pontine nuclei (Koziol et al., 2014; Schmahmann, 2019), supporting the notion that the cerebellum processes information beyond the motor domain.

AVH could arise from changes affecting one or several components of the forward model: the generation of an efference copy (H1), the comparison of expected and actual sensory feedback generating a prediction error (H2), or the updating of the forward model in response to a prediction error (H3). An interface between sensory feedback and control mechanisms (H4) could also contribute to the experience of AVH.

Note. MC = Motor Command; EC = Efference Copy; PE = Prediction Error; UPD = Update; CC = Cognitive Control; PMC = Premotor Cortex; TC = Temporal Cortex; PFC = Prefrontal Cortex; Thal = Thalamus; CER = Cerebellum; SMA = Supplementary Motor Area; IFG = Inferior Frontal Gyrus; STS = Superior Temporal Sulcus; STG = Superior Temporal Gyrus. Blue boxes depict cortical structures; grey boxes depict subcortical structures; black arrows depict a predominant motor pathway; grey arrows depict a predominant sensory pathway. For simplicity, we only illustrate connections between main cortical regions identified in this review, with no further specification.

3. Voices, sensory feedback, and the cerebellum

The human cerebellum is a relatively large structure, containing approximately four times as many neurons as the cerebral cortex and represents about 10 % of the total brain volume (Andersen et al., 1992; Azevedo et al., 2009; Howarth et al., 2010). The cerebellar cortex is characterized by a uniform cytoarchitectonic organization (Ito, 1984), comprising multiple parallel networks that are highly interconnected with the cerebral cortex, the thalamus, and subcortical regions via parallel closed-circuit loops (Buckner et al., 2011; Ito, 2008). Traditionally, the cerebellum has been implicated in sensorimotor control (Manto et al., 2012). However, converging lines of research provide compelling evidence for a role of the cerebellum in higher-order functions such as language and executive functions (Ackermann, 2008; Schmahmann, 2019, 2014, 1996; Schmahmann and Sherman, 1998; Sokolov et al., 2017), as well as emotion (Baumann and Mattingley, 2012; Ivry and Baldo, 1992; Schmahmann and Sherman, 1998). The cerebellum is subdivided into a discrete set of regions that may reflect distinct functional contributions across different tasks (Buckner et al., 2011; King et al., 2019; Stoodley and Schmahmann, 2009). For example, whereas anterior (lobules I–V) and posterior (lobules VIIIa and VIIIb) regions engage in sensorimotor functions (e.g., Grodd et al., 2001), lateral posterior regions (e.g., Crus I, Crus II) are implicated in cognitive and affective functions (Stoodley and Schmahmann, 2009). Specifically, efferents from cerebellar anterior lobe regions are sent to cerebral sensorimotor regions via the interposed nucleus (part of the deep cerebellar nuclei), whereas efferents from the cerebellar posterior lobe are sent to cerebral association areas (e.g., prefrontal cortex) via the ventral dentate (Baumann et al., 2015).

The cerebellum was proposed to instantiate both inverse and forward models (Wolpert et al., 1998), even though the exact computation that it performs remains to be specified (Habas, 2012). Here, we focus on evidence that the cerebellum generates sensory consequences of actions as described by the forward model (Doya, 1999; Ito, 2006; Kawato and Gomi, 1992a). When speaking aloud, the auditory system relies on sensory feedback to distinguish between one's own and other voices. One is also aware of the intention to speak and the act of speaking (Levelt, 2001), i.e. the communicative intention (Wilkinson and Fernyhough, 2017). A forward model may guide perception on the basis of predictions derived from a motor efference copy (Friston, 2005; Schröger et al., 2015). Fig. 1 summarizes hypotheses concerning a possible neural implementation of the forward model. Predicting

sensory feedback to self-generated actions such as speaking requires the concerted effort of several brain regions forming a large-scale network. Besides playing a core role in the implementation of the forward model (Doya, 1999; Ito, 2006; Kawato and Gomi, 1992a), the cerebellum also engages in speech motor control, including the temporal organization of the sound structure when executing speech as well as the temporal organization of inner speech (Ackermann, 2008). Brain imaging studies of speech planning and speech imagery revealed activation of the supplementary motor area (SMA), inferior frontal gyrus, and premotor cortex (Alario et al., 2006), which are all target outputs of the cerebellum (Akkal et al., 2007; Hashimoto et al., 2010; Middleton and Strick, 1997). This activation pattern may reflect the formation of motor commands that are sent to the peripheral nervous system for action execution, including movement of the vocal apparatus.

Neural activity preceding sensory feedback to a motor action (e.g., voice) may reflect how we predict the sensory consequences of action. For example, after voice onset, the cerebellum seems to continuously compare expected and actual sensory feedback (Blakemore et al., 2001; Ito, 2008; Knolle et al., 2013, 2012; Wolpert et al., 1998). Discrepancies between expected and actual sensory feedback are then conveyed via the thalamus to cortical regions including the temporal cortex (e.g., middle and superior temporal gyri; Christoffels et al., 2007; Creutzfeldt et al., 1989; Curio et al., 2000; McGuire et al., 1996; Tourville et al., 2008), premotor, and primary motor cortices (Christoffels et al., 2007; Golfinopoulos et al., 2011; Zheng et al., 2013). Matching of expected and actual sensory feedback occurs approximately within 200 ms post-sound onset (Tian and Poeppel, 2015, 2010) and is reflected in the amplitude modulation of early (Baess et al., 2009) and middle-latency (e.g., Ford et al., 2013) auditory event-related responses (ERP) of the electroencephalogram (EEG). N1 amplitude, peaking around 100 ms post-stimulus onset, is suppressed in response to self-generated sounds, indexing a successful prediction (Baess et al., 2011, 2008; Ford et al., 2001b; Gentsch et al., 2012; Hsu et al., 2016; Hughes et al., 2013; Lange, 2011; Timm et al., 2016). A suppressed N1 amplitude in response to expected sensory feedback seems to mirror reduced reactivity of the auditory cortex to feedback during speaking as observed in fMRI studies (e.g., Christoffels et al., 2007). It seems plausible that the rapid transmission of information via cerebellar-thalamic connections to temporal lobe regions contributes to the sensory suppression effects observed in auditory ERP responses (Knolle et al., 2013, 2012; Schwartze et al., 2012). Specifically, cerebellar-thalamo-cortical connections may contribute to N1 generation by modulating its unspecific subcomponent (Schwartze et al., 2012). The cerebellum receives afferent input from the cerebellum-like dorsal cochlear nucleus (the first relay along the auditory pathway) via the pons early in sound processing (Huang et al., 1982; McLachlan and Wilson, 2017; Petacchi et al., 2005), enabling very short latency responses (Aitkin and Boyd, 1978; McLachlan and Wilson, 2017). Cerebellar responses may thus precede neocortical responses (Huang et al., 1982; Lorenzo et al., 1977a; Misrahy et al., 1961; Shofer and Nahvi, 1969; Wang et al., 1991; Zhang et al., 1990). Direct connectivity of the pons and the lateral cerebellum to afferent pathways of the auditory brainstem as well as spectral integration of auditory input in the pons prior to cerebellar processing (e.g., Aitkin et al., 1975; Aitkin and Boyd, 1978; Snider and Stowell, 1944) makes it likely that a ponto-cerebellar pathway monitors the matching of auditory input with prediction (McLachlan and Wilson, 2017; Schwartze and Kotz, 2013). While evidence documents a cerebellar role in sensory processing, we note that the exact manner in which auditory information is represented in the cerebellum remains unclear (McLachlan and Wilson, 2017).

An error signal arising from a mismatch between expected and actual sensory feedback may trigger online adaptation and motor learning. Less predictable sensory feedback, induced by temporal or content changes, leads to increased activation of the superior temporal gyrus and cerebellar cortex, specifically cerebellar lobules VI (Zheng et al., 2013) and VIII (Pfordresher et al., 2014; Tourville et al., 2008). If sensory feedback is unexpected, updating of the forward model is necessary to optimize adaptive behavior (Brooks et al., 2015; Cullen and Brooks, 2015; Kotz et al., 2014). Evidence further shows that the cerebellum engages in such adaptation of self-generated actions when sensory feedback is changed or less expected (Blakemore et al., 2001; Ishikawa et al., 2016; Ito et al., 1970; Kawato et al., 1987; Miall et al., 1993; Miall and Wolpert, 1996; Wolpert et al., 1998).

Detected discrepancies between expected and actual sensory feedback are transmitted to neocortical target areas, including temporal (Mitra and Snider, 1969; Pastor et al., 2002; Petacchi et al., 2005; Sultan et al., 2012; Teramoto and Snider, 1966; Wolfe, 1972) and frontal (Middleton and Strick, 2001, 1997) areas by means of feedforward connections (Middleton and Strick, 2001, 1997). The cerebellum may thereby influence the updating of the forward model of self-generated action (e.g., self-voice; Fig. 1) through connections that link the cerebellum via the thalamus to premotor areas and selected regions of the prefrontal cortex (Middleton and Strick, 2001, 1997).

In terms of AVH, we need to address whether the same or at least very similar mechanisms apply to the processing of an inner voice, i.e., a voice without an acoustic component. Recent evidence suggests that not only overt speech, conceived as “our most complex motor act” (Levelt, 1989), but also inner speech relies on the forward model (Jack et al., 2019; Scott et al., 2013; Tian et al., 2016; Tian and Poeppel, 2015, 2013, 2012, 2010; Whitford et al., 2017). Speech imagery activates auditory cortex in a similar way as overt speech, indicating that sensory feedback to one’s own actions via a temporally precise and content-specific efference copy occurs even when no sensory feedback is expected (Jack et al., 2019; Tian and Poeppel, 2015, 2010). The responsiveness of the auditory cortex to tones is also reduced when participants silently repeat a statement themselves (Ford et al., 2001b). Estimating the somatosensory consequences of inner speech via an efference copy is thought to provide the perceptual features of the inner voice (Scott, 2013). Such findings suggest that overt and inner speech processing engage similar neurofunctional mechanisms. This, in turn, has far-reaching implications for AVH, which may at least partially arise from misattributions of the source of auditory feedback in general and of inner speech in particular (e.g., Moseley et al., 2013).

In the ensuing sections, we will deconstruct the forward model into its constituent parts and neural correlates from the generation of the efference copy (e.g., the comparison between predicted and actual feedback) to the updating of the forward model in response to unexpected feedback. We emphasize the role of the cerebellum (see Table 1), as the mechanisms by which cerebellar (dys-)function may contribute to AVH are largely obscure. Notwithstanding, we note that our focus on the cerebellum does not exclude the role of other nodes in a network that can explain the experience of hearing a voice in the absence of corresponding acoustic stimulation.

We review the literature suggesting that a dysfunctional cerebellar network may lead to altered sensory feedback and consequently to erratic prediction in AVH. This may also affect higher-level functions such as cognitive control (Dong et al., 2020). Finally, we synthesize evidence on related mechanisms and brain regions that may contribute to an altered sense of agency over one’s own action in AVH, and outline implications of how changes related to the forward model can inform our understanding of AVH.

4. Prediction and processing of sensory feedback to self-voice in AVH

Changes affecting the sensory feedback to the self-voice and potentially also inner speech in AVH may stem from a dysregulation of one or more components of the forward model: the generation of an efference copy, the comparison of expected and actual feedback, or the updating of the forward model in response to a prediction error (Fig. 1). However, generation, comparison, and updating of the model ultimately also engage cognitive control, learning, and monitoring (Ito,

Table 1
Evidence of structural and functional alterations of the cerebellum in voice hearers.

Change	Psychotic AVH
Volume	• Reduced cerebellar grey matter volume [1−2] – specifically in lobules VIIb and VIIIa (pAVH < CTR); lobule VIIIa (pAVH < nAVH) [3]
	• Increased volume of the cerebellar vermis [4]
Connectivity	• Altered bilateral connectivity of DMN, IFG, and CER [5]
	• Increased functional connectivity between salience network and CER [6,7]
	• Decreased effective connectivity from PCC to left CER (pAVH < nAVH) [8]
	• Decreased functional connectivity in left CER (pAVH < CTR; pAVH < nAVH) [9]
	• Decreased functional connectivity between left THAL and right CER [10]
	• Association between a) increased cerebello-cortical resting state connectivity and b) increased FA in the thalamo-motor tract with positive symptom progression in at-risk individuals [11]

Note. The table shows evidence for cerebellar changes in psychotic patients with AVH compared to patients without AVH and/or healthy controls. Studies that documented cerebellar changes in psychotic patients without testing a specific association with positive symptoms, particularly AVH, were not included. Studies probing cerebellar alterations in nonclinical voice hearers are lacking.
CER = Cerebellum; DMN = Default Mode Network; THAL = Thalamus; PCC = Posterior Cingulate Cortex; IFG = Inferior Frontal Gyrus; pAVH = Psychotic Patients with AVH; nAVH = Psychotic Patients without AVH; CTR = Control participants; FA = Fractional Anisotropy.
[1] Huang et al. (2015); [2] Moberget et al. (2019); [3] Cierpka et al. (2017); [4] Levitt et al. (1999); [5] Chang et al. (2015); [6] Mallikarjun et al. (2018); [7] Shin et al. (2015); [8] Zhao et al. (2018); [9] Chen et al. (2019); [10] Clos et al. (2014); [11] Bernard et al. (2017).

2008). Accordingly, dysfunction could arise from an interface between sensory feedback and cognitive control mechanisms in AVH (Fig. 1). To differentiate these components and to specify their relation, we will continue by discussing the available evidence for each of these options.

4.1. An imprecise efference copy?

AVH may result from an imprecise efference copy that prepares the auditory cortex for sensory feedback to the self-voice (Castiello et al., 1991; Goodale et al., 1986). Consistent with the hypothesis that the cerebellum receives the efference copy in parallel to the submission of the commands controlling motor implementation (Green et al., 2007; Requarth and Sawtell, 2014; Tomatsu et al., 2015), cerebellar activity has been found to precede movement onset (Hülsmann et al., 2003), sometimes even before the first change in an electromyographic potential (Anderson and Turner, 1991). Interactions have been identified between the cerebellum and primary motor cortex (M1; Kelly and Strick, 2003; Penhune and Doyon, 2005; Tzvi et al., 2017; Wu et al., 2011), SMA proper, and pre-SMA (Adhikari et al., 2018; Belkhiria et al., 2019; Haggard et al., 1995; Wu et al., 2011). Furthermore, substantiating the role of the cerebellum in response to action preparation and selection (Tachibana et al., 1995; Yamaguchi et al., 1998), lesions in the cerebellar dentate nucleus attenuate the readiness potential, an EEG marker for the preparation of self-initiated movements originating in the SMA proper, pre-SMA, and primary motor cortex (Ikeda et al., 1995; Neshige et al., 1988).

Before hallucination onset, increased activation in the left inferior frontal gyrus, engaged in speech generation, supports the idea that speech motor commands are also emitted in AVH (McGuire et al., 1993; Shergill et al., 2004, 2000). Additionally, increased electromyographic activity in the inferior musculus orbicularis oris (an articulator) was observed during voice hearing (Rapin et al., 2013), revealing that motor simulation without execution and overt speech motor preparation evoke identical activity in AVH (see also Swiney and Sousa, 2014). Such activity is compatible with the generation and transmission of an efference copy (Table 2). After AVH onset, activation extends to regions involved in speech perception, such as the middle and superior temporal gyrus bilaterally (Shergill et al., 2004).

Other patterns of neural activity preceding the onset of sensory feedback may provide further information about the efference copy (Cao et al., 2017). For example, the synchrony of neural oscillations preceding an utterance is reduced in persons with schizophrenia, especially those with severe AVH (Ford et al., 2007b). Pre-speech neural synchrony seems unrelated to N1 amplitude suppression during sensory feedback in psychotic patients, although similar to controls, i.e. it is still higher when speaking than passively listening (Ford et al., 2007b; Table 2). Reduced neural synchrony before an action was not significantly associated with hallucination severity in tasks involving button presses eliciting a pure tone (Ford et al., 2008). The *selective* relationship observed between pre-stimulus neural activity and hallucination severity in tasks involving speech generation may indicate a voice-specific impairment (Pinheiro et al., 2020, 2018). While an efference copy may still be generated, its predictive quality may be reduced due to imprecise or delayed signaling to relevant brain regions such as the cerebellum (e.g., Bernard et al., 2018). In line with this hypothesis, altered connectivity between the inferior frontal gyrus and the cerebellum was found to be a characteristic feature of persons with schizophrenia and AVH but not those without AVH (Chang et al., 2015).

4.2. Insufficient comparison between expected and actual sensory feedback?

The forward model describes how the brain estimates the accuracy of sensory feedback in self-voice production. This feedback is perceived as less salient than external sensations, ultimately leading to suppressed neural activity in sensory cortices. This should not be the case in voice hearers who may fail to predict the sensory consequences of self-voice production.

Support for this hypothesis comes from studies that reported reduced attenuation of auditory cortex activity in response to sensory feedback in psychotic voice hearers (Table 2), particularly in the superior temporal gyrus (Kumari et al., 2010). Similarly, reduced suppression of early auditory ERP responses (N1) was reported in persons with schizophrenia when listening to real-time sensory feedback of their own voice (Ford et al., 2013, 2008, 2007a, 2001a; Ford and Mathalon, 2005, 2004; Heinks-Maldonado et al., 2007; Perez et al., 2012). Specifically, smaller sensory suppression effects were positively correlated with AVH severity (Heinks-Maldonado et al., 2007). Suppression effects in response to inner speech are reported as absent in persons with schizophrenia (Ford et al., 2001c; Ford and Mathalon, 2004). Activity in the left superior temporal gyrus was comparable in psychotic voice hearers when generating overt *vs.* inner speech, whereas it was increased in response to overt *vs.* inner speech in healthy controls (Simons et al., 2010). Such indifference in sensory suppression implies that self and external sensory feedback signals are processed similarly in psychotic voice hearers (Allen et al., 2007b; Ford et al., 2002; Ford and Mathalon, 2004). Consequently, both self-initiated and externally generated sensory stimuli may lead to similarly increased attention (Lange, 2013; Schröger et al., 2015). Furthermore, voice hearers are less sensitive to manipulations (e.g., pitch shifts) of sensory

Table 2
Summary of studies supporting the contribution of the cerebellum to changes in the forward model and higher-level cognitive functions in AVH.

Components of the forward model	Cerebellar contributions	Evidence for preserved mechanism in AVH	Evidence for altered mechanism in AVH
1 Generation of an efference copy	• Attenuated RP after CER lesion [1–2] CER role in movement preparation (e.g., RP) [3]	• Speech motor commands are emitted in AVH [4]	• Altered brain activity preceding feedback onset [5,15,67] [PVH; NCVH [15,67]] • The RP does not predict N1 suppression to sensory feedback [5] [PVH]
2 Comparison between expected and actual sensory feedback to self-voice	• CER contribution to sensory suppression [6–7] • Increased CER activity for sensory prediction errors [8–10] • CER contribution to time discrimination [11–12]	n.a.	• Reduced sensory suppression to self-generated voice feedback and inner speech (STG [13]; N1 [14–17,67]) [PVH; NCVH [15,67]] • Increased CER activation during AVH [18–21] [PVH] • Timing deficits (time discrimination [22]; duration MMN [23]) [PVH]
3 Updating the forward model	• CER role in forward model update and feedforward action control [24–27] • Reduced P3b after CER lesion [28–30]	n.a.	• Persistent external misattribution of self-voice feedback [31–34] [PVH; NCVH [34]] • Reduced P3b to unexpected sounds [35–36] [PVH]
Consequences for higher-level functions	**Cerebellar contributions**	**Evidence for preserved mechanism in AVH**	**Evidence for altered mechanism in AVH**
1 Cognitive control	• Interactions between CER and PFC in cognitive control tasks, such as cognitive inhibition [37–40]	n.a.	• Reduced cognitive inhibition [41–44] [PVH; NCVH [44]] • Enhanced effects of top-down expectations, e.g. in cognitive conflict [45–50] [PVH; NCVH [47,48]]
2 Emotional evaluation of sensory feedback	• CER role in emotional processing and regulation [51] • Interactions between the CER (vermis) and the salience network in emotional processing [52–54]	n.a.	• Increased vermis activation to negative speech [55] [PVH] • Increased AVH frequency after TMS applied to vermis [56] [PVH] • Increased functional connectivity between CER and salience network [57–58] [PVH] • Increased external misattribution errors for negative self-generated speech [32–33,59–60] [PVH]
3 Self-awareness and agency	• Interactions between the CER and the ACC in self-monitoring [61–63]	n.a.	• Altered self-other voice discrimination [31–34] [PVH; NCVH [34]] • Decreased sense of agency over the sensory consequences of one's actions [64–66] [PVH; NCVH [64,66]]

Notes: CER = Cerebellum; PFC = Prefrontal Cortex; STG = Superior Temporal Gyrus; ACC = Anterior Cingulate Cortex; PVH = Psychotic Voice Hearers; NCVH = Non-Clinical Voice Hearers; RP = Readiness Potential; MMN = Mismatch Negativity; PE = Prediction Error; TMS = Transcranial Magnetic Stimulation; n.a. = evidence not available.
The literature search followed the PRISMA guidelines, using the combination of keywords "cerebellum", "hallucinations", "voice", "voice hearing", "psychosis", "schizophrenia", "nonclinical", "feedback".
[1] Neshige et al. (1988); [2] Ikeda et al. (1995); [3] Hülsmann et al. (2003); [4] Rapin et al. (2013); [5] Ford et al. (2008); [6] Knolle et al. (2012); [7] Knolle et al. (2013a); [8] Blakemore and Sirigu (2003); [9] Tseng et al. (2007); [10] Xu-Wilson et al. (2009); [11] Ivry (2000); [12] Blakemore et al. (2001); [13] Kumari et al. (2010); [14] Heinks-Maldonado et al. (2007); [15] Pinheiro et al. (2018); [16] Ford et al. (2001c); [17] Ford and Mathalon (2004); [18] Diederen et al. (2012); [19] Parellada et al. (2008); [20] Sommer et al. (2008); [21] Horga et al. (2011); [22] Waters and Jablensky (2009); [23] Michie (2001); [24] Tseng et al. (2007); [25] Synofzik et al. (2008a); [26] Roth et al. (2013); [27] Butcher et al. (2017); [28] Akshoomoff and Courchesne (1994); [29] Kotz et al. (2014); [30] Tachibana et al. (1995); [31] Allen et al. (2004); [32] Pinheiro et al. (2016); [33] Johns et al. (2010); [34] Allen et al. (2006); [35] Havermans et al. (1999); [36] Higashima et al. (2002); [37] Ramnani (2006); [38] Popa et al. (2017); [39] Liang and Carlson (2019); [40] Schmahmann (2019); [41] Alba-Ferrara et al. (2012); [42] Alba-Ferrara et al. (2013); [43] Pinheiro et al. (2017); [44] Alderson-Day et al. (2019); [45] Haddock et al. (1995); [46] Horga et al. (2014b); [47] Alderson-Day et al. (2017); [48] Powers et al. (2017); [49] Ilankovic et al. (2011); [50] Kambeitz-Ilankovic et al. (2013); [51] Schmahmann et al. (2007); [52] Habas et al. (2009); [53] Snider and Maiti (1976); [54] Zhu et al. (2011); [55] Horga et al. (2014a); [56] Garg et al. (2013); [57] Guo et al. (2015); [58] Shinn et al. (2015); [59] Johns et al. (2001); [60] Costafreda et al. (2008); [61] Schmahmann and Pandya (1997); [62] Schmahmann (1996); [63] Middleton and Strick (1994); [64] Asai and Tanno (2013); [65] Buhler et al. (2016); [66] Sugimori et al. (2011); [67] Pinheiro et al. (2020).

feedback, as seen in reduced suppression effects of early auditory ERP responses (N1) to unmodified (i.e., more predictable) compared to modified (i.e., less predictable) sensory feedback in speaking (Heinks-Maldonado et al., 2007). Sensory feedback to the self-voice is also altered in people with AVH predisposition (Table 2): N1 was larger for self-generated than externally generated voices, whereas it was suppressed for self-generated tones (Pinheiro et al., 2020, 2018). Evidence from psychotic and nonclinical voice hearers thereby suggests that specific alterations of the processing of self-generated vocal sounds may be a core feature of the psychosis continuum (Pinheiro et al., 2020, 2018).

Smaller suppression effects may result from an imprecise comparison of expected and actual sensation, plausibly mediated by the cerebellum: accordingly, the N1 was found to respond sensitively to cerebellar dysfunction (Knolle et al., 2013, 2012). The cerebellum is well suited to perform a fast computation of sensory feedback and related prediction errors (e.g., Miall and Wolpert, 1996). Cerebellar responses were found to precede neocortical responses, even in primary sensory areas (Lorenzo et al., 1977b), potentially via a direct sensory afferent pathway between the cerebellum and the dorsal cochlear nucleus (Wang et al., 1991). The cerebellum is sensitive to relatively abrupt changes in sensory input occurring roughly within 10 – 30 ms (e.g., Apps et al., 1995), conveyed to the thalamus and then transmitted to cortical regions (reviewed in Schwartze and Kotz, 2013) such as the primary auditory cortex (Mitra and Snider, 1969; Pastor et al., 2002; Petacchi et al., 2005; Sultan et al., 2012; Teramoto and Snider, 1966; Wolfe, 1972). A comparison of expected and actual sensory feedback may occur in the deep cerebellar nuclei (Neshige et al., 1988; Streng et al., 2018). Specifically, sensory error signals are encoded in complex spike discharges of Purkinje cells (Gilbert and Thach, 1977; Ito, 2013, 2000; Kawato and Gomi, 1992b; Kitazawa et al., 1998; Stone and Lisberger, 1986). However, as sensory systems are organized hierarchically (e.g., Baldeweg, 2007), the difference between expected and actual sensory feedback may be propagated to other hierarchical levels such as the prefrontal cortex (Middleton and Strick, 2001, 1997) and anterior cingulate cortex (Anand et al., 1959; Snider and Maiti, 1976).

Cerebellar activity correlates with sensory prediction errors. In healthy individuals, cerebellar activity is reduced in response to self-generated (*vs.* external) sensory feedback (Blakemore et al., 1999; Blakemore and Sirigu, 2003; Brooks and Cullen, 2013), but increased when there is a discrepancy between an action goal and the sensory consequences of that action (Blakemore and Sirigu, 2003). Moreover, persons with cerebellar disorders fail to use prediction errors during motor learning (Tseng et al., 2007; Xu-Wilson et al., 2009). Suggesting imprecise sensory feedback, hallucinated voices are associated with increased cerebellar activation in symptom-capture studies (Diederen et al., 2012; Parellada et al., 2008; Sommer et al., 2008; Table 2), which correlates with the emotional content of AVH (Horga et al., 2011). This increased activation indicates that the cerebellum responds to a discrepancy between expected and actual sensory voice feedback.

A delay in generating an efference copy or its transmission (Koreki et al., 2015) also results in a mismatch between expected and actual sensory feedback. Supporting the hypothesis of a delayed efference copy, Whitford and collaborators (Whitford et al., 2011) found that by imposing a 50 ms delay between an action (button press) and a resulting tone, the N1 suppression effect was comparable in psychotic persons and healthy controls. Structural and functional changes in the cerebellum and cerebellar-thalamo-cortical networks can also explain the lack of motor-induced sensory suppression, considering that cerebellar activity is modulated by the magnitude of temporal deviations between an action and sensory feedback (Blakemore et al., 2001) and temporally constrained to approximately one second (Ivry and Keele, 1989). Dysfunction of cerebellar-thalamo-cortical circuitry has been related to the pathophysiology of psychosis (e.g., Clos et al., 2014; Table 1) as well as decreased cerebellar-cortical resting state connectivity (Bottmer et al., 2005; Collin et al., 2011; Deicken et al., 2001; Greenstein et al., 2011; Heath et al., 1979; Ichimiya et al., 2001; Keller et al., 2003; Kim et al., 2014; Loeber et al., 2001; Lohr and Jeste, 1986; Maloku et al., 2010; Okugawa et al., 2003; Schmitt et al., 2011; Shinn et al., 2015). This is the case in psychotic patients reporting AVH (Chang et al., 2015) and selectively associated with positive (but not negative) symptom progression in at-risk individuals within a year of psychosis onset (Bernard et al., 2017). Cerebellar-thalamo-cortical network development and connectivity at baseline assessment (particularly connectivity between lobule V and motor cortex) in at-risk adolescents predicted positive symptom progression over 12 months (Bernard et al., 2017) and may be indicative of abnormal sensorimotor integration and failure to attribute the source of sensory feedback. Cerebellar abnormalities found in at-risk individuals (Bernard et al., 2014; Dean et al., 2014; Pantelis et al., 2003) were consequently proposed as a biomarker of disease progression (Bernard et al., 2017). In addition, impairments of time discrimination, typically observed in patients with cerebellar pathology, are also observed in psychotic voice hearers (Waters and Jablensky, 2009; Table 2). Experiments probing the mismatch negativity (MMN, an ERP component peaking at 100 – 200 ms after change onset – Näätänen et al., 2007) link sensory prediction mechanisms (lesion data – Moberget et al., 2008; Restuccia et al., 2007; cerebellar transcranial direct current stimulation – Chen et al., 2014), and specifically the generation of precisely timed expectancies regarding forthcoming sensory information (Ivry, 2000; Moberget et al., 2008) to the cerebellum. These studies show that the MMN response to duration deviants is strongly affected in persons with schizophrenia (Michie, 2001). The longer the delay between an action and sensory feedback, the less agency of one's own action is perceived (Gentsch et al., 2012; Timm et al., 2016), and the more salient feedback is perceived. Accordingly, delayed transmission of the efference copy could contribute to a reduced sense of agency.

4.3. Erroneous updating of the forward model?

Sensory prediction errors likely drive adaptation in the forward model. The updating of the forward model relates to a temporally specific adaption process that relies on the detection of discrepancies. Unexpected changes of sensory feedback lead to increased activation of the cerebellum compared to expected feedback (Blakemore et al., 2001), consistent with the role of the cerebellum in error-based learning (Doya, 1999; Herzfeld et al., 2018; Popa et al., 2016; Popa and Ebner, 2019; reviewed in Johnson et al., 2019). Connections from the dorsal cochlear nucleus to the cerebellum may provide direct sensory input to large-scale cortico-cerebellar networks engaged in the generation and adaptation of the forward model (Schwartze and Kotz, 2013). The error signals would then allow the cerebellum to issue a feedforward command to frontal lobe regions via the thalamus for sensorimotor control (Ramnani, 2006). The basal ganglia could further modulate the sensitivity of the cerebellum to sensory prediction errors, by estimating the behavioral value of the new state (Ohmae and Medina, 2015). If the forward model provides accurate predictions, automatic small-scale adaptation to unexpected changes may be implemented without considerable delay, sparing neocortical neural and cognitive resources (e.g., attention). However, if a prediction error disproportionally increases, the forward model is either not updated or updated inadequately. For example, if feedback differs fundamentally from the prediction, the error signal becomes unreliable (Katseff et al., 2012). Consequently, flexible and rapid updating does not ensue, and feedback may be treated as externally generated (Friston, 2012).

Cerebellar lesions result in impaired updating of predictions of sensory feedback (Butcher et al., 2017; Roth et al., 2013; Synofzik et al., 2008a; Tseng et al., 2007). Hence, the forward model is not appropriately fine-tuned and updated over time, whereas errors will continue. Consequently, feedforward control of action might be compromised. Comparable to cerebellar patients, who display a reduced capability to compensate for variability in motor behavior (e.g.,

dysmetric saccades), manipulations of sensory feedback to action may be perceived as unreliable by voice hearers, leading the motor system to disregard altered feedback rather than to implement corrections or update the forward model.

Behavioral studies showed that psychotic voice hearers as well as individuals at high risk for developing psychosis (Johns et al., 2010) more likely attribute increased acoustic ambiguity or uncertainty in sensory feedback to external rather than internal sources (e.g., pitch-distorted voice feedback – Allen et al., 2004; Pinheiro et al., 2016; Table 2). A tendency to misperceive a pitch-shifted (Allen et al., 2006) or shorter (Pinheiro et al., 2019) self-generated than other voice is also found in nonclinical voice hearers with high AVH proneness (Table 2). EEG studies have documented erroneous updating of the forward model in AVH. EEG responses in the P300 range (an ERP peaking approximately 300 ms after stimulus onset – Polich, 2007) are thought to reflect the updating of a mental model of the environment when unexpected input is detected (Kotz et al., 2014). Changes in P3b amplitude (decreased amplitude – Akshoomoff and Courchesne, 1994; Kotz et al., 2014) and latency (increased latency – Tachibana et al., 1995) were reported in cerebellar patients. Similarly, P3b amplitude reductions to unexpected task-relevant tones were enhanced in psychotic patients with AVH (Havermans et al., 1999; Higashima et al., 2003; Table 2).

5. Higher-level cognitive functions

5.1. Changes in cognitive control?

The cerebellum may not only mediate the comparison of expected and actual sensory feedback but also regulate cognitive function through interactions with neocortical regions (Liang and Carlson, 2019; Popa et al., 2017; Ramnani, 2006; Schmahmann, 2019). Interactions of the postero-lateral cerebellum (e.g., Crus I, Crus II) and the prefrontal cortex (Schmahmann and Pandya, 1997) form the basis of cerebellar involvement in higher-level processes such as cognitive control, a hallmark of executive functions (Ide and Li, 2011; Ramnani, 2006). Intact cerebellar processing reduces cognitive load, facilitates executive control, and optimizes performance in tasks engaging attentional processes (Ghajar and Ivry, 2009; Khilkevich et al., 2018), particularly executive attention (Habas et al., 2009; Mannarelli et al., 2019; Manto and Jissendi, 2012). Accordingly, focal cerebellar lesions have been associated with impaired conflict-related cognitive processing (Schweizer et al., 2007).

Sensory feedback to one's own action reduces cognitive load by attenuating sensory processing, which is perceived as less surprising and salient. Consequently, cognitive resources might be spared for the processing of less predictable external stimuli. Changes related to specific components in the forward model, namely the comparison between expected and actual feedback, may affect this asymmetry. If sensory feedback to the self-voice is treated the same way as external stimuli, resource allocation may be symmetric and require overall higher levels of attention to differentiate the sources. This would increase cognitive load and events in the environment might be perceived as more demanding and less controllable. If the forward model is not continuously fine-tuned, performance becomes more effortful and less automatic, and thus more heavily reliant on prefrontal cortical function (Ito, 2008).

In line with this idea, alterations in cerebellar sensorimotor circuitry were proposed to partly account for high-level cognitive cerebellar dysfunction in persons with schizophrenia, based on evidence for increased resting-state functional connectivity between the cerebellar sensorimotor and cerebellar cognitive networks (Dong et al., 2020): higher cognitive effort is thought to be employed in response to excessive error input. Decreased cortico-cerebellar connectivity with Crus I and Crus II was also found in these patients (Repovs et al., 2011; Table 1), which suggests less efficient neural signaling implicated in cognitive control (Bernard and Mittal, 2015). Moreover, volumetric changes in cognitive regions of the cerebellum (specifically reduced grey matter volume in lobules VIIb and VIIIa) are associated with persistent positive symptoms of psychosis such as AVH (Cierpka et al., 2017; Table 2). Cerebellar dysfunction could also influence the basal ganglia via a disynaptic pathway through the thalamus and thereby contribute to reduced cognitive control (Bostan et al., 2013; Prevosto and Sommer, 2013).

Attention gates the influence of sensory information on perception and can be directed internally to coordinate thoughts, memories, and emotions (Miller, 2000). Inhibition is one of the executive functions of attention: it might be required to withhold irrelevant sensory information or responses that are not appropriate given a specific context (Miller, 2000). Altered executive attention could explain the inability to inhibit hallucinated voices once they occur or to ignore emotionally salient features of hallucinated voices (Alba-Ferrara et al., 2013, 2012; Pinheiro et al., 2017). Along the same lines, intentional cognitive inhibition has been associated with hallucination proneness (Alderson-Day et al., 2019). Reduced cognitive control could also contribute to enhancing expectation effects as a result of inadequately coded prediction errors (Table 2). Attentional demands could be increased when voice hearers discriminate salient social and environmental stimuli and make them more vulnerable to top-down expectations, i.e., the knowledge derived from prior experience rather than from sensory stimulation (Miller, 2000). This is evident in the increased tendency of voice hearers to report acoustic changes in acoustically undistorted speech (Haddock et al., 1995) and speech percepts in signal detection tasks (Horga et al., 2014b) or degraded auditory stimuli (Alderson-Day et al., 2017). Both psychotic and nonclinical voice hearers were found to report the presence of a (absent) 1-kHz tone after learning its association with a visual stimulus via classical conditioning (Powers et al., 2017) or to misattribute the source of sensory feedback when it was preceded by the picture of another person's face (Ilankovic et al., 2011; Kambeitz-Ilankovic et al., 2013). A voice percept may also be assigned social significance depending on prior expectations (Griffin and Fletcher, 2017). Such findings are consistent with the hypothesis that both enhanced sensation and increased sensitivity to top-down expectations may account for AVH (Corlett et al., 2019).

5.2. Altered emotional quality of sensory feedback to self-voice?

The forward model provides a comprehensive framework to explain misattribution of external sensory feedback in AVH. However, it does not explain why hallucinated voices often have an emotional quality (Larøi et al., 2012; McCarthy-Jones et al., 2014b; Nayani and David, 1996). The cerebellum not only engages in the control of action, but contributes to non-motor functions, which may rely on the same computational mechanisms (D'Angelo et al., 2011). For example, the cerebellum participates in emotional processing and regulation (Schmahmann et al., 2007), enabled via connections from the fastigial nucleus to the amygdala, hippocampus, and middle temporal gyri (Heath and Harper, 1974). Accordingly, cerebellar stimulation elicits responses in limbic regions, including the amygdala and the cingulate cortex (e.g., Anand et al., 1959). Cerebellar lobule VI interacts with the salience network to prioritize the processing of emotionally relevant stimuli in a context-dependent manner (Habas et al., 2009). Furthermore, the cerebellar vermis and the amygdala interact during emotional associative processes (Snider and Maiti, 1976; Zhu et al., 2011). One of the central functions of the cerebellum in emotional processing could therefore be the integration of emotional cues and their transmission to the cerebral cortex for behavioral adaptation (Adamaszek et al., 2016).

It is well established that focal cerebellar lesions can give rise to cognitive and affective disruptions, defining the cognitive-affective cerebellar syndrome (Schmahmann and Sherman, 1998), which mirrors some clinical criteria of psychosis. Disruption of cerebellar activity via Transcranial Magnetic Stimulation (TMS) resulted in impaired

categorization of emotional faces, in both explicit and incidental emotional processing (Ferrari et al., 2018). Whereas the cerebellum engaged in the processing of both positive and negative emotional information, a predominant role for negative emotions was suggested (Adamaszek et al., 2014; Annoni et al., 2003; Clausi et al., 2015; Diano et al., 2016; Ferrucci et al., 2012; Lupo et al., 2015; Maschke et al., 2000; Park et al., 2010; Zhu et al., 2007).

In line with the presumed contribution of the cerebellum to the emotional quality of hallucinated voices, the cerebellar vermis was found to be more active in response to aversive sentences mimicking the typical features of AVH (Horga et al., 2014a; Table 2). Similarly, repetitive TMS applied to the vermis resulted in increased frequency of AVH (Garg et al., 2013). Further evidence comes from studies reporting increased volume of the cerebellar vermis (Levitt et al., 1999), altered connectivity in cortico-cerebellar-thalamo-cortical circuits (De La Iglesia-Vaya et al., 2014) as well as increased resting-state functional connectivity between the cerebellum and salience, ventral attention, and default-mode networks in psychotic voice hearers (Guo et al., 2015; Mallikarjun et al., 2018; Shinn et al., 2015). The cerebellar vermis may thus represent an interface between sensory stimulation (via a cortico-pontine-cerebellar circuit), the emotional state of the participant, and the motor response (Adamaszek et al., 2016). Due to altered cerebellar function and connectivity, the absence of an appropriate prediction of the emotional qualities of inner speech mediated by the cerebellum may make such input seem unintended or external. As the cerebellum is thought to be specifically engaged in explicit self-monitoring of negative voice cues (Adamaszek et al., 2016), altered cerebellar function could further contribute to external misattribution errors for negative self-generated speech in self-monitoring tasks at least in psychotic voice hearers (Costafreda et al., 2008; Johns et al., 2010, 2001; Pinheiro et al., 2016). Emotional manipulations may not be perceived as one's own voice anymore but rather as external ('*this is not my voice*') and negative.

5.3. Altered self-awareness and agency?

Sensory feedback to one's own action is critical for an undeterred sense of agency. The forward model provides a plausible explanation for the perceptual integration of action-based sensory feedback and the differentiation of self-generated (highly predictable) and external (less predictable) sensory input. The less predictable and salient a sensory event is, the more likely it is perceived as reflecting an externally generated event. Cognitive evaluation may be engaged to determine whether a given stimulus is self-generated or produced by another agent (Synofzik et al., 2008b). For example, despite the detection of an error resulting from a delay between an action and sensory feedback, individuals may still attribute this feedback to their own action or perceive themselves as agents of an action (e.g., Farrer et al., 2013).

Alterations in prefrontal and anterior cingulate cortices, involved in error processing, may contribute to failed source monitoring of self-voice feedback in AVH (Clos et al., 2014) and to errors in self-voice recognition (e.g., Costafreda et al., 2008; Johns et al., 2010, 2001; Pinheiro et al., 2019, 2016). Studies comparing resting-state functional connectivity in psychotic voice hearers and controls showed that the predisposition to experience AVH may be associated with the decoupling of voice production (e.g., involving the SMA) and self-monitoring (involving dorsolateral prefrontal cortex, ventrolateral prefrontal cortex, anterior cingulate, inferior parietal cortex) systems (Clos et al., 2014). The inferior frontal gyrus is part of a self-awareness network that regulates a sense of agency over an action (David et al., 2008; Farrer et al., 2004; Jardri et al., 2007; Leube et al., 2003; Nahab et al., 2011). In AVH, the increased perceived reality of the hallucinated voices is associated with increased activation of the inferior frontal gyrus (Raij et al., 2009).

Studies on inner speech have highlighted the role of the anterior cingulate cortex in dysfunctional self-monitoring in psychotic voice hearers (Shergill et al., 2000). The anterior cingulate cortex is part of a performance monitoring network (a critical function of cognitive control – Shenhav et al., 2013) that is active when an individual becomes aware of a prediction error (Orr and Hester, 2012) and may contribute to an altered sense of agency (Table 2). It monitors cognitive demands and regulates the allocation of cognitive resources (Shenhav et al., 2013). The anterior cingulate cortex has feedforward projections to (Schmahmann, 1996; Schmahmann and Pandya, 1997) and feedback projections from the cerebellum (Middleton and Strick, 1994). During self-other voice discrimination (including both distorted self- and non-self-voice feedback), psychotic voice hearers did not activate the anterior cingulate cortex to the same extent as controls and non-hallucinating psychotic patients: the dorsal anterior cingulate cortex was more active in response to unchanged than to acoustically distorted self-speech, whereas the opposite was observed in healthy controls and non-hallucinating patients (Allen et al., 2007a). Anterior cingulate cortex activation patterns in AVH suggest increased cognitive demands associated with the processing of more predictable (*vs.* less predictable) feedback, as well as reduced error awareness with manipulated feedback. Consequently, an error correction response is not initiated. Furthermore, altered effective connectivity between the left superior temporal and anterior cingulate cortex is associated with the tendency to perceive one's own voice as generated by another person (Mechelli et al., 2007). Altered function of the anterior cingulate cortex may hence contribute to a decreased sense of agency over the sensory consequences of one's own actions (Asai and Tanno, 2013; Bühler et al., 2016; Sugimori et al., 2011). Accordingly, the self-generated voice would be evaluated as external ('this is not my voice') in voice hearers (Allen et al., 2006, 2004; Brébion et al., 2016; Johns et al., 2010; Pinheiro et al., 2016).

6. A mechanistic framework of AVH

The evidence discussed in the preceding sections suggests that changes of specific components within the forward model are key to a better understanding of AVH. Together with changes in cortical and thalamic structures, alterations in the cerebellum may give rise to the experience of reduced ownership over one's own voice and, consequently to AVH (Fig. 2).

Processing changes affecting the comparison of expected and actual sensory feedback (Fig. 1, H2) as well as the updating of the forward model (Fig. 1, H3) are strongly supported by the existing literature (Fig. 2, Panels 2 and 3). Although one study reported alterations in efference copy generation (Ford et al., 2007b), the majority of the reviewed evidence suggests that this mechanism is preserved in voice hearers (Fig. 2, Panel 1; Table 2). However, even though motor areas may compile an efference copy in voice hearers, this copy seems to either not contain and/or relay accurate (e.g., temporal) information regarding sensory feedback (Fig. 2, Panel 1). Consequently, a prediction error might ensue when comparing expected and actual sensory feedback, i.e. the processing of the self-generated voice is changed (Fig. 2, Panel 2). Further, due to altered error sensitivity, the forward model may not be adequately updated (Fig. 2, Panel 3): feedback is consequently perceived as input of external origin, leading to a reduced sense of agency. Changes in components of the forward model while speaking (namely, comparison between expected and actual sensory feedback to the self-voice; updating) could lead to a failure in distinguishing internal states and external events, setting the stage for AVH. Considering the putative role of the cerebellum also in the generation and modulation of inner speech (Ait Khelifa-Gallois et al., 2015; Marvel and Desmond, 2010) plausibly via sub-vocal articulation (Chen and Desmond, 2005; Marvel and Desmond, 2012), mental events (e.g., inner speech) might be misrecognized as generated by an external agent (Jones and Fernyhough, 2007), even in the absence of an overt action.

Dysfunction in the circuitry putatively implied in the forward model might produce alterations in sensory feedback by changing its effects in

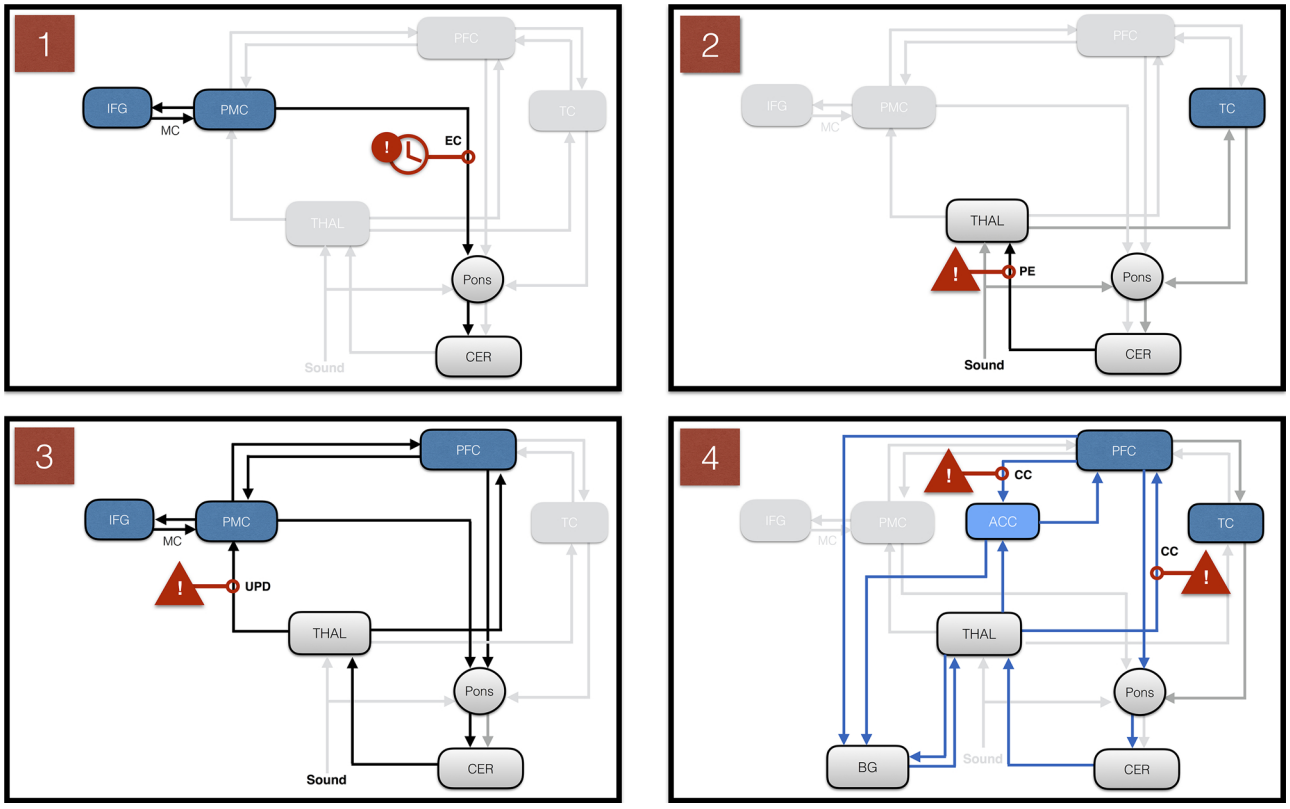

Fig. 2. A working model of the cerebellar role in AVH.
Fig. 2 adapts Fig. 1 to illustrate each of the formulated hypotheses on mechanistic changes in the forward model in AVH. AVH likely result in structural (at least in psychotic voice hearers) and functional (in both psychotic and nonclinical voice hearers) changes, affecting the comparison of expected and actual sensory feedback (Panel 2) and the updating of the forward model (Panel 3), engaging cerebellar circuitry. Higher-level cognitive dysfunctions (e.g., cognitive control; emotional evaluation) likely result from an interaction with these altered components of the forward model (Panel 4; blue line) and contribute to the experience of AVH. A large body of evidence supports this hypothesis (see also Table 2).
Note. MC = Motor Command; EC = Efference Copy; PE = Prediction Error; UPD = Update; CC = Cognitive Control; IFG = Inferior Frontal Gyrus; PMC = Primary Motor Cortex; PFC = Prefrontal Cortex; ACC = Anterior Cingulate Cortex; TC = Temporal Cortex; THAL = Thalamus; CER = Cerebellum; BG = Basal Ganglia. Blue boxes depict cortical structures; grey boxes depict subcortical structures; light blue boxes depict structures within the medial PFC; black arrows depict the motor pathway; grey arrows depict the sensory pathway; blue arrows depict circuits engaged in cognitive functions such as cognitive control, which may overlap with motor circuits. The red triangle illustrates malfunctioning components of the forward model or the interface with dysfunctional higher-level cognitive functions.

distant structures within the circuitry. Indeed, a likely possibility is that all areas of the cerebral cortex that project to the cerebellum are targets of cerebellar output (Caligiore et al., 2017). For example, cerebellar dysfunction could result in a failure to effectively communicate with neocortical network nodes, including the prefrontal cortex, thereby contributing to ineffective cognitive control and altered emotional processing of sensory feedback to the voice (Fig. 2, Panel 4). This may contribute to explicit evaluations of sensory feedback as alien and to an overall reduced sense of control over events in the environment.

Of note is that the phenomenology of AVH is heterogeneous and not one of the described mechanisms alone accounts for the full experience of AVH (McCarthy-Jones et al., 2014a; Stephane, 2013). Further, most of the reviewed studies (Fig. 2; Table 2) tested persons diagnosed with psychotic disorders, which are often clinically heterogeneous (e.g., co-occurrence of negative symptoms or of other positive symptoms such as delusions) and present additional confounds, namely the effects of medication and hospitalization. Specifically, antipsychotic medication may bias conclusions about brain regions with dopaminergic innervation such as the basal ganglia and also to some degree the cerebellum (Giompres and Delis, 2005; Hurley et al., 2003; Ikai et al., 1992; Melchitzky and Lewis, 2000; Panagopoulos et al., 1991). Studies involving nonclinical voice hearers are hence warranted as they provide an appropriate model for investigating AVH in isolation (Kelleher and Cannon, 2011). There are strong reasons to expect that the way in which distinct components of the forward model may be changed differentiates between nonclinical and psychotic voice hearers and, consequently, contributes to the phenomenological diversity of 'hearing voices'. For example, more pronounced changes in the cerebellar vermis and/or in the connectivity of the cerebellum with limbic regions is expected in psychotic AVH, which are often described as threatening and derogatory, but less so in nonclinical AVH (e.g., Larøi et al., 2012; McCarthy-Jones et al., 2014b; Nayani and David, 1996). Changes involving higher-level cognitive functions, such as a stronger impact of cognitive control, are expected when AVH result in significant functional impairment, which is the case in psychotic voice hearers (Waters and Fernyhough, 2017). A major challenge will be to unravel whether and why different mechanisms may culminate in voice rather than visual hallucinations or in other types of auditory hallucinations. In particular, voice-specific rather than generalized, altered sensory feedback may explain why hallucinated voices represent the most common atypical perceptual experience (Pinheiro et al., 2020, 2018).

Acknowledging that the picture is far from complete, the framework developed here offers a novel perspective and starting point for future research. Deeper insights into how cerebellar dysfunction contributes to AVH will depend on asking more refined questions. First, the exact nature of the operations and processes relying on the cerebellum remains elusive. A uniform cytoarchitecture, exquisite temporal precision, and extensive reciprocal connections with neocortical regions are especially well-suited for cerebellar operations in relation to the forward model (Sokolov et al., 2017). Nonetheless, it remains to be

specified whether the role of the cerebellum in AVH is primary, secondary, or compensatory (Bareš et al., 2019). For instance, altered cerebellar function could represent a secondary consequence of basal ganglia dysfunction: the cerebellum works in concert with the basal ganglia via a disynaptic projection through the thalamus (Caligiore et al., 2017); further, the function of the basal ganglia is heavily dependent on dopamine neuromodulation (Smith and Villalba, 2008), which is known to be dysregulated in psychosis (Winton-Brown et al., 2014). Moreover, as there are distinct functional regions in the cerebellum (Witter and De Zeeuw, 2015; see also the MOdular Selection And Identification Controller [MOSAIC] computational model – Imamizu et al., 2003; Wolpert and Ghahramani, 2000), a more specific analysis of the fine-grained functional topography within the cerebellum would likely provide additional specification of how it contributes to the experience of AVH. It is possible that different cerebellar zones contribute to distinct operations underpinning the forward model (Johnson et al., 2019; Stoodley and Schmahmann, 2009, 2010). Most advanced methods for the analysis of brain imaging data are optimized for the cerebrum, but not the cerebellum, which may be related to a cortico-centric bias in neuroscience research (Parvizi, 2009). As such, the involvement of the cerebellum in AVH is possibly underreported.

Finally, although the current review focuses on data from psychotic and nonclinical voice hearers, it is worth noting that AVH can also be present in other psychiatric conditions (e.g., bipolar disorder – Toh et al., 2015) and in neurological disorders (e.g., epilepsy – Serino et al., 2014). Elucidating the neurofunctional mechanisms underlying sensory feedback in voice production in AVH across different disorders will enhance our understanding of whether alterations in these mechanisms represent a neural substrate specific to AVH rather than psychosis. This knowledge would be critical for a precise diagnosis and for the development of more effective intervention strategies.

7. Conclusions and future directions

Following the review of the existing evidence, we can identify additional directions for further examination. Fundamental issues can be addressed by rigorously testing theory-based predictions using various neuroimaging techniques:

1 *Situational factors*: The role of state variables in the forward model has been discussed in motor control theory (Frith et al., 2000). AVH in psychotic patients are often elicited by negative emotional or physical (e.g., tiredness) states (Luhrmann et al., 2019; Ratcliffe and Wilkinson, 2016), and typically elicit an emotional (often fear) response (Peters et al., 2012). Hallucinatory predisposition in nonclinical individuals is associated with higher levels of anxiety and self-focus (Allen et al., 2005). Some AVH may arise from hypersensitivity to auditory threat (Dudley et al., 2014). Hence, an individual's affect may represent a perpetuating factor of AVH in psychosis. Reciprocal connections between the cerebellum and brainstem rely on neurotransmitters involved in mood regulation (e.g., serotonin, dopamine – Dempesy et al., 1983; Marcinkiewicz et al., 1989), suggesting that the cerebellum (particularly, the vermis) plays a role in the regulation of emotions (Adamaszek et al., 2016). The role of these connections in AVH elicitation and maintenance remains to be clarified.

2 *Salience and emotion*: Future studies should determine how the forward model links to resting state networks such as the salience network to produce specific phenomenological features of AVH along the psychosis continuum (e.g., Alderson-Day et al., 2015). For example, AVH typically have a more negative quality in psychotic than in nonclinical voice hearers (Baumeister et al., 2017). Further, it is not clear whether and how voice salience and emotion are associated with distinct types of sensory feedback and relate to the forward model differently.

3 *Sensory suppression across sensory modalities*: Self-initiated stimuli across different sensory modalities may give rise to differences in sensory suppression patterns (Mifsud and Whitford, 2017). It is important to understand whether altered sensory suppression in response to self-initiated voices in AVH is specific to voices, enhanced for voices compared to other types of sounds, and whether these alterations extend to different sensory modalities (e.g., vision). Such differentiation may not only be reflected in neocortical responses but also in patterns of cerebellar activation, which may be modulated by the modality of sensory feedback (Johnson et al., 2019).

4 *Voice hearing along the psychosis continuum*: It remains to be clarified whether alterations in the forward model are similar in psychotic and non-psychotic voice hearers. Most of the studies tested psychotic patients with AVH, whereas studies probing the forward model and cerebellar changes in nonclinical persons with AVH remain scarce. Future studies should examine whether distinct components of the forward model (Fig. 2) relate to changes along the psychosis continuum and whether such changes could predict psychosis onset.

The studies discussed in the current review support the contribution of neural mechanistic changes in relation to the forward model in AVH, which are at least partly associated with cerebellar dysfunction. The reviewed evidence lays the foundation for further discussion and provides a tentative answer to the key question why some people hear voices in the absence of corresponding acoustic input. At the neural level, the answer likely resides in studying the function of the cerebellum from an integrative perspective that considers its interplay with the cortex, thalamus, and basal ganglia.

Acknowledgments

This work was supported by Fundação para a Ciência e a Tecnologia (FCT; grant number PTDC/MHC-PCN/0101/2014). The authors would also like to thank two anonymous reviewers for their constructive comments and valuable suggestions.

References

Ackermann, H., 2008. Cerebellar contributions to speech production and speech perception: psycholinguistic and neurobiological perspectives. Trends Neurosci. 31, 265–272. https://doi.org/10.1016/j.tins.2008.02.011.

Adamaszek, M., D'Agata, F., Kirkby, K.C., Trenner, M.U., Sehm, B., Steele, C.J., Berneiser, J., Strecker, K., 2014. Impairment of emotional facial expression and prosody discrimination due to ischemic cerebellar lesions. Cerebellum 13, 338–345. https://doi.org/10.1007/s12311-013-0537-0.

Adamaszek, M., D'Agata, F., Ferrucci, R., Habas, C., Keulen, S., Kirkby, K.C., Leggio, M., Marien, P., Molinari, M., Moulton, E., Orsi, L., Van Overwalle, F., Papadelis, C., Priori, A., Sacchetti, B., Schutter, D.J., Styliadis, C., Verhoeven, J., 2016. Consensus paper: cerebellum and emotion. Cerebellum 16, 552–576. https://doi.org/10.1007/s12311-016-0815-8.

Adhikari, B.M., Epstein, C.M., Dhamala, M., 2018. Enhanced brain network activity in complex movement rhythms: a simultaneous fMRI-EEG study. Brain Connect. 8, 68–81. https://doi.org/10.1089/brain.2017.0547.

Ait Khelifa-Gallois, N., Puget, S., Longaud, A., Laroussinie, F., Soria, C., Sainte-Rose, C., Dellatolas, G., 2015. Clinical evidence of the role of the cerebellum in the suppression of overt articulatory movements during reading. A study of reading in children and adolescents treated for cerebellar Pilocytic Astrocytoma. Cerebellum 14 (2), 97–105. https://doi.org/10.1007/s12311-014-0612-1.

Aitkin, L.M., Boyd, J., 1978. Acoustic input to the lateral pontine nuclei. Hear. Res. 1, 67–77. https://doi.org/10.1016/0378-5955(78)90010-2.

Aitkin, L.M., Boyd, J., Aitkin, M., 1975. Responses of single units in cerebellar vermis of the cat to monaural and binaural stimuli. J. Neurophysiol. 38, 418–429.

Akkal, D., Dum, R.P., Strick, P.L., 2007. Supplementary motor area and presupplementary motor area: targets of basal ganglia and cerebellar output. J. Neurosci. 27, 10659–10673. https://doi.org/10.1523/jneurosci.3134-07.2007.

Akshoomoff, N.A., Courchesne, E., 1994. ERP evidence for a shifting attention deficit in patients with damage to the cerebellum. J. Cogn. Neurosci. 6, 388–399. https://doi.org/10.1162/jocn.1994.6.4.388.

Alario, F.X., Chainay, H., Lehericy, S., Cohen, L., 2006. The role of the supplementary motor area (SMA) in word production. Brain Res. 1076, 129–143. https://doi.org/10.1016/j.brainres.2005.11.104.

Alba-Ferrara, L., Fernyhough, C., Weis, S., Mitchell, R.L.C., Hausmann, M., 2012. Contributions of emotional prosody comprehension deficits to the formation of

auditory verbal hallucinations in schizophrenia. Clin. Psychol. Rev. 32, 244–250.
Alba-Ferrara, L., de Erausquin, G.A., Hirnstein, M., Weis, S., Hausmann, M., 2013. Emotional prosody modulates attention in schizophrenia patients with hallucinations. Front. Hum. Neurosci. 7, 59. https://doi.org/10.3389/fnhum.2013.00059.
Alderson-Day, B., McCarthy-Jones, S., Fernyhough, C., 2015. Hearing voices in the resting brain: a review of intrinsic functional connectivity research on auditory verbal hallucinations. Neurosci. Biobehav. Rev. 55, 78–87. https://doi.org/10.1016/j.neubiorev.2015.04.016.
Alderson-Day, B., Lima, C.F., Evans, S., Krishnan, S., Shanmugalingam, P., Fernyhough, C., Scott, S.K., 2017. Distinct processing of ambiguous speech in people with non-clinical auditory verbal hallucinations. Brain 9, 2475–2489. https://doi.org/10.1093/brain/awx206.
Alderson-Day, B., Smailes, D., Moffatt, J., Mitrenga, K., Moseley, P., Fernyhough, C., 2019. Intentional inhibition but not source memory is related to hallucination-proneness and intrusive thoughts in a university sample. Cortex 113, 267–278. https://doi.org/10.1016/j.cortex.2018.12.020.
Aleman, A., Böcker, K.B.E., Hijman, R., Kahn, R.S., De Haan, E.H.F., 2002. Hallucinations in schizophrenia: imbalance between imagery and perception? Schizophr. Res. 57, 315–316. https://doi.org/10.1016/S0920-9964(01)00291-2.
Allen, P., Johns, L.C., Fu, C.H.Y., Broome, M.R., Vythelingum, G.N., McGuire, P.K., 2004. Misattribution of external speech in patients with hallucinations and delusions. Schizophr. Res. 69, 277–287. https://doi.org/10.1016/j.schres.2003.09.008.
Allen, P., Freeman, D., McGuire, P.K., Garety, P.A., Kuipers, E., Fowler, D.G., Bebbington, P.E., Green, C., Dunn, G., Ray, K., 2005. The prediction of hallucinatory predisposition in non-clinical individuals: examining the contribution of emotion and reasoning. Br. J. Clin. Psychol. 44, 127–132. https://doi.org/10.1348/014466504X20044.
Allen, P., Freeman, D., Johns, L., McGuire, P., 2006. Misattribution of self-generated speech in relation to hallucinatory proneness and delusional ideation in healthy volunteers. Schizophr. Res. 84, 281–288. https://doi.org/10.1016/j.schres.2006.01.021.
Allen, P., Aleman, A., McGuire, P.K., 2007a. Inner speech models of auditory verbal hallucinations: evidence from behavioural and neuroimaging studies. Int. Rev. Psychiatry 19, 407–415. https://doi.org/10.1080/09540260701486498.
Allen, P., Amaro, E., Fu, C.H.Y., Williams, S.C.R., Brammer, M.J., Johns, L.C., McGuire, P.K., 2007b. Neural correlates of the misattribution of speech in schizophrenia. Br. J. Psychiatry 190, 162–169. https://doi.org/10.1192/bjp.bp.106.025700.
Alonso-Solís, A., Vives-Gilabert, Y., Grasa, E., Portella, M.J., Rabella, M., Sauras, R.B., Roldán, A., Núñez-Marín, F., Gómez-Ansón, B., Pérez, V., Alvarez, E., Corripio, I., 2015. Resting-state functional connectivity alterations in the default network of schizophrenia patients with persistent auditory verbal hallucinations. Schizophr. Res. https://doi.org/10.1016/j.schres.2014.10.047.
Anand, B.K., Malhotra, C.L., Singh, B., Dua, S., 1959. Cerebellar projections to limbic system. J. Neurophysiol. 22, 451–457.
Andersen, B.B., Korbo, L., Pakkenberg, B., 1992. A quantitative study of the human cerebellum with unbiased stereological techniques. J. Comp. Neurol. 326, 549–560. https://doi.org/10.1002/cne.903260405.
Anderson, M.E., Turner, R.S., 1991. Activity of neurons in cerebellar-receiving and pallidal-receiving areas of the thalamus of the behaving monkey. J. Neurophysiol. 66, 879–893. https://doi.org/10.1152/jn.1991.66.3.879.
Andreasen, N.C., Pierson, R., 2008. The role of the cerebellum in schizophrenia. Biol. Psychiatry 64, 81–88. https://doi.org/10.1016/j.biopsych.2008.01.003.
Andreasen, N.C., O'Leary, D.S., Cizadlo, T., Arndt, S., Rezai, K., Ponto, L.L., Watkins, G.L., Hichwa, R.D., 1996. Schizophrenia and cognitive dysmetria: a positron-emission tomography study of dysfunctional prefrontal-thalamic-cerebellar circuitry. Proc. Natl. Acad. Sci. https://doi.org/10.1073/pnas.93.18.9985.
Andreasen, N.C., Paradiso, S., O'Leary, D.S., 1998. "Cognitive dysmetria" as an integrative theory of schizophrenia: A dysfunction in cortical-subcortical-cerebellar circuitry? Schizophr. Bull. 24, 203–218. https://doi.org/10.1093/oxfordjournals.schbul.a033321.
Andreasen, N.C., Nopoulos, P., O'Leary, D.S., Miller, D.D., Wassink, T., Flaum, M., 1999. Defining the phenotype of schizophrenia: cognitive dysmetria and its neural mechanisms. Biol. Psychiatryhttps://doi.org/10.1016/S0006-3223(99)00152-3.
Annoni, J.M., Ptak, R., Caldara-Schnetzer, A.S., Khateb, A., Pollermann, B.Z., 2003. Decoupling of autonomic and cognitive emotional reactions after cerebellar stroke. Ann. Neurol. 53, 654–658. https://doi.org/10.1002/ana.10549.
Apps, R., Hartell, N.A., Armstrong, D.M., 1995. Step phase-related excitability changes in spino-olivocerebellar paths to the c1 and c3 zones in cat cerebellum. J. Physiol. (Paris) 483, 687–702. https://doi.org/10.1113/jphysiol.1995.sp020614.
Asai, T., Tanno, Y., 2013. Why must we attribute our own action to ourselves? Auditory hallucination like-experiences as the results both from the explicit self-other attribution and implicit regulation in speech. Psychiatry Res. 207, 179–188. https://doi.org/10.1016/j.psychres.2012.09.055.
Azevedo, F.A.C., Carvalho, L.R.B., Grinberg, L.T., Farfel, J.M., Ferretti, R.E.L., Leite, R.E.P., Filho, W.J., Lent, R., Herculano-Houzel, S., 2009. Equal numbers of neuronal and nonneuronal cells make the human brain an isometrically scaled-up primate brain. J. Comp. Neurol. 513, 532–541. https://doi.org/10.1002/cne.21974.
Baess, P., Jacobsen, T., Schröger, E., 2008. Suppression of the auditory N1 event-related potential component with unpredictable self-initiated tones: evidence for internal forward models with dynamic stimulation. Int. J. Psychophysiol. 70, 137–143. https://doi.org/10.1016/j.ijpsycho.2008.06.005.
Baess, P., Widmann, A., Roye, A., Schröger, E., Jacobsen, T., 2009. Attenuated human auditory middle latency response and evoked 40-Hz response to self-initiated sounds. Eur. J. Neurosci. 29, 1514–1521. https://doi.org/10.1111/j.1460-9568.2009.06683.x.
Baess, P., Horvath, J., Jacobsen, T., Schroger, E., 2011. Selective suppression of self-initiated sounds in an auditory stream: an ERP study. Psychophysiology 48, 1276–1283. https://doi.org/10.1111/j.1469-8986.2011.01196.x.
Baldeweg, T., 2007. ERP repetition effects and mismatch negativity generation: A predictive coding perspective. J. Psychophysiol. 21, 204–213. https://doi.org/10.1027/0269-8803.21.34.204.
Banse, R., Scherer, K.R., 1996. Acoustic profiles in vocal emotion expression. J. Pers. Soc. Psychol. 70, 614–636. https://doi.org/10.1037/0022-3514.70.3.614.
Bareš, M., Apps, R., Avanzino, L., Breska, A., D'Angelo, E., Filip, P., Gerwig, M., Ivry, R.B., Lawrenson, C.L., Louis, E.D., Lusk, N.A., Manto, M., Meck, W.H., Mitoma, H., Petter, E.A., 2019. Consensus paper: decoding the contributions of the cerebellum as a time machine. From neurons to clinical applications. Cerebellum 18, 266–286. https://doi.org/10.1007/s12311-018-0979-5.
Barkus, E., Stirling, J., Hopkins, R., McKie, S., Lewis, S., 2007. Cognitive and neural processes in non-clinical auditory hallucinations. Br. J. Psychiatry 51, s76–81. https://doi.org/10.1192/bjp.191.51.s76.
Bastian, A.J., 2006. Learning to predict the future: the cerebellum adapts feedforward movement control. Curr. Opin. Neurobiol. 16, 645–649. https://doi.org/10.1016/j.conb.2006.08.016.
Bauer, S.M., Schanda, H., Karakula, H., Olajossy-Hilkesberger, L., Rudaleviciene, P., Okribelashvili, N., Chaudhry, H.R., Idemudia, S.E., Gscheider, S., Ritter, K., Stompe, T., 2011. Culture and the prevalence of hallucinations in schizophrenia. Compr. Psychiatry 52, 319–325. https://doi.org/10.1016/j.comppsych.2010.06.008.
Baumann, O., Mattingley, J.B., 2012. Functional topography of primary emotion processing in the human cerebellum. Neuroimage 61, 805–811. https://doi.org/10.1016/j.neuroimage.2012.03.044.
Baumann, O., Borra, R.J., Bower, J.M., Cullen, K.E., Habas, C., Ivry, R.B., Leggio, M., Mattingley, J.B., Molinari, M., Moulton, E.A., Paulin, M.G., Pavlova, M.A., Schmahmann, J.D., Sokolov, A.A., 2015. Consensus paper: the role of the cerebellum in perceptual processes. Cerebellum 14, 197–220. https://doi.org/10.1007/s12311-014-0627-7.
Baumeister, D., Sedgwick, O., Howes, O., Peters, E., 2017. Auditory verbal hallucinations and continuum models of psychosis: a systematic review of the healthy voice-hearer literature. Clin. Psychol. Rev. 51, 125–141. https://doi.org/10.1016/j.cpr.2016.10.010.
Beavan, V., 2011. Towards a definition of "hearing voices": a phenomenological approach. Psychosis 3, 63–73. https://doi.org/10.1080/17522431003615622.
Beavan, V., Read, J., Cartwright, C., 2011. The prevalence of voice-hearers in the general population: a literature review. J. Ment. Heal. 20, 281–292. https://doi.org/10.3109/09638237.2011.562262.
Belin, P., Fecteau, S., Bédard, C., 2004. Thinking the voice: neural correlates of voice perception. Trends Cogn. Sci. (Regul. Ed.) 8, 129–135. https://doi.org/10.1016/j.tics.2004.01.008.
Belkhiria, C., Mssedi, E., Habas, C., Driss, T., de Marco, G., 2019. Collaboration of cerebello-rubral and cerebello-striatal loops in a motor preparation task. Cerebellum 18, 203–211. https://doi.org/10.1007/s12311-018-0980-z.
Bell, V., 2013. A community of one: social cognition and auditory verbal hallucinations PLoS Biol. 11. https://doi.org/10.1371/journal.pbio.1001723.
Bentall, R.P., 1990. The Illusion of Reality: A Review and Integration of Psychological Research on Hallucinations. Psychol. Bull. 107, 82–95. https://doi.org/10.1037/0033-2909.107.1.82.
Bernard, J.A., Mittal, V.A., 2015. Dysfunctional activation of the cerebellum in schizophrenia: a functional neuroimaging meta-analysis. Clin. Psychol. Sci. 3, 545–566. https://doi.org/10.1177/2167702614542463.
Bernard, J.A., Dean, D.J., Kent, J.S., Orr, J.M., Pelletier-Baldelli, A., Lunsford-Avery, J.R., Gupta, T., Mittal, V.A., 2014. Cerebellar networks in individuals at ultra high-risk of psychosis: impact on postural sway and symptom severity. Hum. Brain Mapp. 35, 4064–4078. https://doi.org/10.1002/hbm.22458.
Bernard, J.A., Orr, J.M., Mittal, V.A., 2017. Cerebello-thalamo-cortical networks predict positive symptom progression in individuals at ultra-high risk for psychosis. Neuroimage Clin. 14, 622–628. https://doi.org/10.1016/j.nicl.2017.03.001.
Bernard, J.A., Orr, J.M., Dean, D.J., Mittal, V.A., 2018. The cerebellum and learning of non-motor associations in individuals at clinical-high risk for psychosis. Neuroimage Clin. 19, 137–146. https://doi.org/10.1016/j.nicl.2018.03.023.
Bielawski, M., Bondurant, H., 2015. Psychosis following a stroke to the cerebellum and midbrain: a case report. Cerebellum Ataxias 2, 17. https://doi.org/10.1186/s40673-015-0037-8.
Birchwood, M., Meaden, A., Trower, P., Gilbert, P., Plaistow, J., 2000. The power and omnipotence of voices: subordination and entrapment by voices and significant others. Psychol. Med. (Paris) 30, 337–344. https://doi.org/10.1017/S0033291799001828.
Blakemore, S.J., Frith, C., 2003. Self-awareness and action. Curr. Opin. Neurobiol. 13, 219–224. https://doi.org/10.1016/S0959-4388(03)00043-6.
Blakemore, S.J., Sirigu, A., 2003. Action prediction in the cerebellum and in the parietal lobe. Exp. Brain Res. 153, 239–245. https://doi.org/10.1007/s00221-003-1597-z.
Blakemore, S.J., Frith, C.D., Wolpert, D.M., 1999. Spatio-temporal prediction modulates the perception of self-produced stimuli. J. Cogn. Neurosci. 11, 551–559. https://doi.org/10.1162/089892999563607.
Blakemore, S.J., Smith, J., Steel, R., Johnstone, C.E., Frith, C.D., 2000. The perception of self-produced sensory stimuli in patients with auditory hallucinations and passivity experiences: evidence for a breakdown in self-monitoring. Psychol. Med. (Paris) 30, 1131–1139. https://doi.org/10.1017/S0033291799002676.
Blakemore, S.-J., Frith, C.D., Wolpert, D.M., 2001. The cerebellum is involved in predicting the sensory consequences of action. Neuroreport 12, 1879–1884. https://doi.org/10.1097/00001756-200107030-00023.
Bostan, A.C., Dum, R.P., Strick, P.L., 2013. Cerebellar networks with the cerebral cortex and basal ganglia. Trends Cogn. Sci. (Regul. Ed.) 17, 241–254. https://doi.org/10.

1016/j.tics.2013.03.003.
Bottmer, C., Bachmann, S., Pantel, J., Essig, M., Amann, M., Schad, L.R., Magnotta, V., Schröder, J., 2005. Reduced cerebellar volume and neurological soft signs in first-episode schizophrenia. Psychiatry Res. - Neuroimaging 140, 239–250. https://doi.org/10.1016/j.pscychresns.2005.02.011.
Brébion, G., Smith, M.J., Gorman, J.M., Amador, X., 1996. Reality monitoring failure in schizophrenia: the role of selective attention Schizophr. Res. 22, 173–180. https://doi.org/10.1016/S0920-9964(96)00054-0.
Brébion, G., Amador, X., David, A., Malaspina, D., Sharif, Z., Gorman, J.M., 2000. Positive symptomatology and source-monitoring failure in schizophrenia - an analysis of symptom-specific effects. Psychiatry Res. 95, 119–131. https://doi.org/10.1016/S0165-1781(00)00174-8.
Brébion, G., Stephan-Otto, C., Ochoa, S., Roca, M., Nieto, L., Usall, J., 2016. Impaired self-monitoring of inner speech in schizophrenia patients with verbal hallucinations and in non-clinical individuals prone to hallucinations. Front. Psychol. 7, 1381. https://doi.org/10.3389/fpsyg.2016.01381.
Brodal, P., 1978. The corticopontine projection in the rhesus monkey origin and principles of organization. Brain 101, 251–283. https://doi.org/10.1093/brain/101.2.251.
Brooks, J.X., Cullen, K.E., 2013. The primate cerebellum selectively encodes unexpected self-motion. Curr. Biol. 23, 947–955. https://doi.org/10.1016/j.cub.2013.04.029.
Brooks, J.X., Carriot, J., Cullen, K.E., 2015. Learning to expect the unexpected: rapid updating in primate cerebellum during voluntary self-motion. Nat. Neurosci. 18, 1310–1317. https://doi.org/10.1038/nn.4077.
Buckner, R.L., Krienen, F.M., Castellanos, A., Diaz, J.C., Yeo, B.T.T., 2011. The organization of the human cerebellum estimated by intrinsic functional connectivity. J. Neurophysiol. 106, 2322–2345. https://doi.org/10.1152/jn.00339.2011.
Bühler, T., Kindler, J., Schneider, R.C., Strik, W., Dierks, T., Hubl, D., Koenig, T., 2016. Disturbances of agency and ownership in schizophrenia: an auditory verbal event related potentials study. Brain Topogr. 29, 716–727. https://doi.org/10.1007/s10548-016-0495-1.
Butcher, P.A., Ivry, R.B., Kuo, S.-H., Rydz, D., Krakauer, J.W., Taylor, J.A., 2017. The cerebellum does more than sensory-prediction-error-based learning in sensorimotor adaptation tasks. J. Neurophysiol. 118, 1622–1636. https://doi.org/10.1152/jn.00451.2017.
Caligiore, D., Pezzulo, G., Baldassarre, G., Bostan, A.C., Strick, P.L., Doya, K., Helmich, R.C., Dirkx, M., Houk, J., Jörntell, H., Lago-Rodriguez, A., Galea, J.M., Miall, R.C., Popa, T., Kishore, A., Verschure, P.F.M.J., Zucca, R., Herreros, I., 2017. Consensus paper: towards a systems-level view of cerebellar function: the interplay between cerebellum, basal ganglia, and cortex Cerebellum 16, 203–229. https://doi.org/10.1007/s12311-016-0763-3.
Cao, H., Cannon, T.D., 2019. Cerebellar dysfunction and schizophrenia: from "cognitive dysmetria" to a potential therapeutic target. Am. J. Psychiatry 176, 498–500. https://doi.org/10.1176/appi.ajp.2019.19050480.
Cao, L., Thut, G., Gross, J., 2017. The role of brain oscillations in predicting self-generated sounds. Neuroimage 147, 895–903. https://doi.org/10.1016/j.neuroimage.2016.11.001.
Cao, H., Chén, O.Y., Chung, Y., Forsyth, J.K., McEwen, S.C., Gee, D.G., Bearden, C.E., Addington, J., Goodyear, B., Cadenhead, K.S., Mirzakhanian, H., Cornblatt, B.A., Carrión, R.E., Mathalon, D.H., McGlashan, T.H., Perkins, D.O., Belger, A., Seidman, L.J., Thermenos, H., Tsuang, M.T., van Erp, T.G.M., Walker, E.F., Hamann, S., Anticevic, A., Woods, S.W., Cannon, T.D., 2018. Cerebello-thalamo-cortical hyperconnectivity as a state-independent functional neural signature for psychosis prediction and characterization. Nat. Commun. 9, 1–9. https://doi.org/10.1038/s41467-018-06350-7.
Castiello, U., Paulignan, Y., Jeannerod, M., 1991. Temporal dissociation of motor responses and subjective awareness a study in normal subjects. Brain 114, 2639–2655. https://doi.org/10.1093/brain/114.6.2639.
Chang, X., Xi, Y.-B., Cui, L.-B., Wang, H.-N., Sun, J.-B., Zhu, Y.-Q., Huang, P., Collin, G., Liu, K., Xi, M., Qi, S., Tan, Q.-R., Miao, D.-M., Yin, H., 2015. Distinct inter-hemispheric dysconnectivity in schizophrenia patients with and without auditory verbal hallucinations. Sci. Rep. 5, 11218. https://doi.org/10.1038/srep11218.
Chen, S.H.A., Desmond, J.E., 2005. Cerebrocerebellar networks during articulatory rehearsal and verbal working memory tasks. Neuroimage 24, 332–338. https://doi.org/10.1016/j.neuroimage.2004.08.032.
Chen, J.C., Hämmerer, D., D'Ostilio, K., Casula, E.P., Marshall, L., Tsai, C.H., Rothwell, J.C., Edwards, M.J., 2014. Bi-directional modulation of somatosensory mismatch negativity with transcranial direct current stimulation: an event related potential study. J. Physiol. (Paris) 592, 745–757. https://doi.org/10.1113/jphysiol.2013.260331.
Chen, X., Ji, G.J., Zhu, C., Bai, X., Wang, L., He, K., Gao, Y., Tao, L., Yu, F., Tian, Y., Wang, K., 2019. Neural correlates of auditory verbal hallucinations in schizophrenia and the therapeutic response to theta-burst transcranial magnetic stimulation. Schizophr. Bull. 45, 474–483. https://doi.org/10.1093/schbul/sby054.
Christoffels, I.K., Formisano, E., Schiller, N.O., 2007. Neural correlates of verbal feedback processing: an fMRI study employing overt speech. Hum. Brain Mapp. 28, 868–879. https://doi.org/10.1002/hbm.20315.
Cierpka, M., Wolf, N.D., Kubera, K.M., Schmitgen, M.M., Vasic, N., Frasch, K., Wolf, R.C., 2017. Cerebellar contributions to persistent auditory verbal hallucinations in patients with schizophrenia. Cerebellum 16, 964–972. https://doi.org/10.1007/s12311-017-0874-5.
Clausi, S., Coricelli, G., Pisotta, I., Pavone, E.F., Lauriola, M., Molinari, M., Leggio, M., 2015. Cerebellar damage impairs the self-rating of regret feeling in a gambling task. Front. Behav. Neurosci. 9, 113. https://doi.org/10.3389/fnbeh.2015.00113.
Clos, M., Diederen, K.M.J., Meijering, A.L., Sommer, I.E., Eickhoff, S.B., 2014. Aberrant connectivity of areas for decoding degraded speech in patients with auditory verbal hallucinations. Brain Struct. Funct. 219, 581–594. https://doi.org/10.1007/s00429-013-0519-5.
Collin, G., HulshoffPol, H.E., Haijma, S.V., Cahn, W., Kahn, R.S., van den Heuvel, M.P., 2011. Impaired cerebellar functional connectivity in schizophrenia patients and their healthy siblings. Front. Psychiatry 2, 73. https://doi.org/10.3389/fpsyt.2011.00073.
Copolov, D.L., Mackinnon, A., Trauer, T., 2004. Correlates of the affective impact of auditory hallucinations in psychotic disorders. Schizophr. Bull. 30, 163–171. https://doi.org/10.1093/oxfordjournals.schbul.a007060.
Corlett, P.R., Horga, G., Fletcher, P.C., Alderson-Day, B., Schmack, K., Powers III, A.R., 2019. Hallucinations and strong priors. Trends Cogn. Sci. (Regul. Ed.) 23, 114–127. https://doi.org/10.1016/j.tics.2018.12.001.
Costafreda, S.G., Brébion, G., Allen, P., McGuire, P.K., Fu, C.H.Y., 2008. Affective modulation of external misattribution bias in source monitoring in schizophrenia. Psychol. Med. (Paris) 38, 821–824. https://doi.org/10.1017/S0033291708003243.
Creutzfeldt, O., Ojemann, G., Lettich, E., 1989. Neuronal activity in the human lateral temporal lobe II. Responses to the subjects own voice. Exp. Brain Res. 77, 476–489. https://doi.org/10.1007/BF00249600.
Cullen, K.E., Brooks, J.X., 2015. Neural correlates of sensory prediction errors in monkeys: evidence for internal models of voluntary self-motion in the cerebellum. Cerebellum 14, 31–34. https://doi.org/10.1007/s12311-014-0608-x.
Curio, G., Neuloh, G., Numminen, J., Jousmäki, V., Hari, R., 2000. Speaking modifies voice-evoked activity in the human auditory cortex. Hum. Brain Mapp. 9, 183–191 doi:10.1002/(SICI)1097-0193(200004)9:4 < 183::AID-HBM1 > 3.0.CO;2-Z.
D'Angelo, E., Mazzarello, P., Prestori, F., Mapelli, J., Solinas, S., Lombardo, P., Cesana, E., Gandolfi, D., Congi, L., 2011. The cerebellar network: from structure to function and dynamics. Brain Res. Rev. 66, 5–15. https://doi.org/10.1016/j.brainresrev.2010.10.002.
Daalman, K., van Zandvoort, M., Bootsman, F., Boks, M., Kahn, R., Sommer, I., 2011. Auditory verbal hallucinations and cognitive functioning in healthy individuals. Schizophr. Res. 132, 203–207. https://doi.org/10.1016/j.schres.2011.07.013.
David, N., Newen, A., Vogeley, K., 2008. The "sense of agency" and its underlying cognitive and neural mechanisms. Conscious. Cogn. 17, 523–534. https://doi.org/10.1016/j.concog.2008.03.004.
De La Iglesia-Vaya, M., Escartí, M.J., Molina-Mateo, J., Martí-Bonmatí, L., Gadea, M., Castellanos, F.X., Aguilar García-Iturrospe, E.J., Robles, M., Biswal, B.B., Sanjuan, J., 2014. Abnormal synchrony and effective connectivity in patients with schizophrenia and auditory hallucinations. Neuroimage Clin. 6, 171–179. https://doi.org/10.1016/j.nicl.2014.08.027.
Dean, D.J., Bernard, J.A., Orr, J.M., Pelletier-Baldelli, A., Gupta, T., Carol, E.E., Mittal, V.A., 2014. Cerebellar morphology and procedural learning impairment in neuroleptic-naive youth at ultrahigh risk of psychosis. Clin. Psychol. Sci. 2, 152–164. https://doi.org/10.1177/2167702613500039.
Deicken, R.F., Feiwell, R., Schuff, N., Soher, B., 2001. Evidence for altered cerebellar vermis neuronal integrity in schizophrenia. Psychiatry Res. 107, 125–134.
Dempesy, C.W., Tootle, D.M., Fontana, C.J., Fitzjarrell, A.T., Garey, R.E., Heath, R.G., 1983. Stimulation of the paleocerebellar cortex of the cat: increased rate of synthesis and release of catecholamines at limbic sites. Biol. Psychiatry 18, 127–132.
Diano, M., D'Agata, F., Cauda, F., Costa, T., Geda, E., Sacco, K., Duca, S., Torta, D.M., Geminiani, G.C., 2016. Cerebellar clustering and functional connectivity during pain processing. Cerebellum 15, 343–356. https://doi.org/10.1007/s12311-015-0706-4.
Diederen, K.M.J., Neggers, S.F.W., Daalman, K., Blom, J.D., Goekoop, R., Kahn, R.S., Sommer, I.E.C., 2010. Deactivation of the parahippocampal gyrus preceding auditory hallucinations in schizophrenia. Am. J. Psychiatry 167, 427–435. https://doi.org/10.1176/appi.ajp.2009.09040456.
Diederen, K.M.J., Daalman, K., De Weijer, A.D., Neggers, S.F.W., Van Gastel, W., Blom, J.D., Kahn, R.S., Sommer, I.E.C., 2012. Auditory hallucinations elicit similar brain activation in psychotic and nonpsychotic individuals. Schizophr. Bull. 38, 1074–1082. https://doi.org/10.1093/schbul/sbr033.
Diederen, K.M., Neggers, S.F., de Weijer, A.D., van Lutterveld, R., Daalman, K., Eickhoff, S.B., Clos, M., Kahn, R.S., Sommer, I.E., 2013. Aberrant resting-state connectivity in non-psychotic individuals with auditory hallucinations. Psychol. Med. (Paris) 43, 1685–1696. https://doi.org/10.1017/S0033291712002541.
Dong, D., Luo, C., Guell, X., Wang, Y., He, H., Duan, M., Eickhoff, S.B., Yao, D., 2020. Compression of cerebellar functional gradients in schizophrenia. Schizophr. Bull. https://doi.org/10.1093/schbul/sbaa016.
Doya, K., 1999. What are the computations of the cerebellum, the basal ganglia and the cerebral cortex? Neural Netw. 12, 961–974. https://doi.org/10.1016/S0893-6080(99)00046-5.
Dudley, R., Dodgson, G., Sarll, G., Halhead, R., Bolas, H., McCarthy-Jones, S., 2014. The effect of arousal on auditory threat detection and the relationship to auditory hallucinations. J. Behav. Ther. Exp. Psychiatry 45, 311–318. https://doi.org/10.1016/j.jbtep.2014.02.002.
Farrer, C., Franck, N., Frith, C.D., Decety, J., Georgieff, N., D'Amato, T., Jeannerod, M., 2004. Neural correlates of action attribution in schizophrenia. Psychiatry Res. - Neuroimaging 131, 31–44. https://doi.org/10.1016/j.pscychresns.2004.02.004.
Farrer, C., Valentin, G., Hupé, J.M., 2013. The time windows of the sense of agency. Conscious. Cogn. 22, 1431–1441. https://doi.org/10.1016/j.concog.2013.09.010.
Feinberg, I., 1978. Efference copy and corollary discharge: implications for thinking and its disorders*. Schizophr. Bull. 4, 636–640. https://doi.org/10.1093/schbul/4.4.636.
Fernyhough, C., 2004. Alien voices and inner dialogue: towards a developmental account of auditory verbal hallucinations. New Ideas Psychol. 22, 49–68. https://doi.org/10.1016/j.newideapsych.2004.09.001.
Ferrari, C., Oldrati, V., Gallucci, M., Vecchi, T., Cattaneo, Z., 2018. The role of the cerebellum in explicit and incidental processing of facial emotional expressions: a study with transcranial magnetic stimulation. Neuroimage 169, 256–264. https://doi.org/10.1016/j.neuroimage.2017.12.026.
Ferrucci, R., Giannicola, G., Rosa, M., Fumagalli, M., Boggio, P.S., Hallett, M., Zago, S.,

Priori, A., 2012. Cerebellum and processing of negative facial emotions: cerebellar transcranial DC stimulation specifically enhances the emotional recognition of facial anger and sadness. Cogn. Emot. 26, 786–799. https://doi.org/10.1080/02699931.2011.619520.
Fletcher, P.C., Frith, C.D., 2009. Perceiving is believing: a Bayesian approach to explaining the positive symptoms of schizophrenia. Nat. Rev. Neurosci. 10, 48–58. https://doi.org/10.1038/nrn2536.
Ford, J.M., Mathalon, D.H., 2004. Electrophysiological evidence of corollary discharge dysfunction in schizophrenia during talking and thinking. J. Psychiatr. Res. 38, 37–46. https://doi.org/10.1016/S0022-3956(03)00095-5.
Ford, J.M., Mathalon, D.H., 2005. Corollary discharge dysfunction in schizophrenia: can it explain auditory hallucinations? Int. J. Psychophysiol. 58, 179–189. https://doi.org/10.1016/j.ijpsycho.2005.01.014.
Ford, J.M., Mathalon, D.H., Heinks, T., Kalba, S., Faustman, W.O., Roth, W.T., 2001a. Neurophysiological evidence of corollary discharge dysfunction in schizophrenia. Am. J. Psychiatry 158, 2069–2071. https://doi.org/10.1176/appi.ajp.158.12.2069.
Ford, J.M., Mathalon, D.H., Kalba, S., Whitfield, S., Faustman, W.O., Roth, W.T., 2001b. Cortical responsiveness during inner speech in schizophrenia: an event-related potential study. Am. J. Psychiatry 158, 1914–1916. https://doi.org/10.1176/appi.ajp.158.11.1914.
Ford, J.M., Mathalon, D.H., Kalba, S., Whitfield, S., Faustman, W.O., Roth, W.T., 2001c. Cortical responsiveness during talking and listening in schizophrenia: an event-related brain potential study. Biol. Psychiatry 50, 540–549. https://doi.org/10.1016/S0006-3223(01)01166-0.
Ford, J.M., Mathalon, D.H., Whitfield, S., Faustman, W.O., Roth, W.T., 2002. Reduced communication between frontal and temporal lobes during talking in schizophrenia. Biol. Psychiatry 51, 485–492. https://doi.org/10.1016/S0006-3223(01)01335-X.
Ford, J.M., Gray, M., Faustman, W.O., Roach, B.J., Mathalon, D.H., 2007a. Dissecting corollary discharge dysfunction in schizophrenia. Psychophysiology 44, 522–529. https://doi.org/10.1111/j.1469-8986.2007.00533.x.
Ford, J.M., Roach, B.J., Faustman, W.O., Mathalon, D.H., 2007b. Synch before you speak: auditory hallucinations in schizophrenia. Am. J. Psychiatry 164, 458–466. https://doi.org/10.1176/appi.ajp.164.3.458.
Ford, J.M., Roach, B.J., Faustman, W.O., Mathalon, D.H., 2008. Out-of-Synch and out-of-Sorts: dysfunction of motor-sensory communication in schizophrenia Biol. Psychiatry 63, 736–743. https://doi.org/10.1016/j.biopsych.2007.09.013.
Ford, J.M., Mathalon, D.H., Roach, B.J., Keedy, S.K., Reilly, J.L., Gershon, E.S., Sweeney, J.A., 2013. Neurophysiological evidence of corollary discharge function during vocalization in psychotic patients and their nonpsychotic first-degree relatives. Schizophr. Bull. 39, 1272–1280. https://doi.org/10.1093/schbul/sbs129.
Friston, K., 2005. A theory of cortical responses. Philos. Trans. R. Soc. Lond., B, Biol. Sci. 360, 815–836. https://doi.org/10.1098/rstb.2005.1622.
Friston, K., 2012. Prediction, perception and agency. Int. J. Psychophysiol. 83, 248–252. https://doi.org/10.1016/j.ijpsycho.2011.11.014.
Frith, C.D., Done, D.J., 1988. Towards a neuropsychology of schizophrenia. Br. J. Psychiatry 153, 437–443. https://doi.org/10.1192/bjp.153.4.437.
Frith, C., Rees, G., Friston, K., 1998. Psychosis and the experience of self: brain systems underlying self-monitoring. Ann. N. Y. Acad. Sci. 843, 170–178. https://doi.org/10.1111/j.1749-6632.1998.tb08213.x.
Frith, C.D., Blakemore, S.-J., Wolpert, D.M., 2000. Abnormalities in the awareness and control of action. Philos. Trans. R. Soc. B Biol. Sci. 355, 1771–1788. https://doi.org/10.1098/rstb.2000.0734.
Garg, S., Goyal, N., Tikka, S.K., Sinha, V.K., 2013. Exacerbation of auditory verbal hallucinations with adjunctive high-frequency cerebellar vermal repetitive transcranial magnetic stimulation in schizophrenia: a case report. J. ECT 29, 65–66. https://doi.org/10.1097/YCT.0b013e3182706aa1.
Gentsch, A., Schütz-Bosbach, S., 2011. I did it: unconscious expectation of sensory consequences modulates the experience of self-agency and its functional signature. J. Cogn. Neurosci. 23, 3817–3828. https://doi.org/10.1162/jocn_a_00012.
Gentsch, A., Kathmann, N., Schütz-Bosbach, S., 2012. Reliability of sensory predictions determines the experience of self-agency. Behav. Brain Res. 228, 415–422. https://doi.org/10.1016/j.bbr.2011.12.029.
Ghajar, J., Ivry, R.B., 2009. The predictive brain state: Asynchrony in disorders of attention? Neuroscientist 15, 232–242. https://doi.org/10.1177/1073858408326429.
Gilbert, P.F.C., Thach, W.T., 1977. Purkinje cell activity during motor learning. Brain Res. 128, 309–328. https://doi.org/10.1016/0006-8993(77)90997-0.
Giompres, P., Delis, F., 2005. Dopamine transporters in the cerebellum of mutant mice. Cerebellum 4, 105–111. https://doi.org/10.1080/14734220510007851.
Golfinopoulos, E., Tourville, J.A., Bohland, J.W., Ghosh, S.S., Nieto-Castanon, A., Guenther, F.H., 2011. FMRI investigation of unexpected somatosensory feedback perturbation during speech. Neuroimage 55, 1324–1338. https://doi.org/10.1016/j.neuroimage.2010.12.065.
Goodale, M.A., Pelisson, D., Prablanc, C., 1986. Large adjustments in visually guided reaching do not depend on vision of the hand or perception of target displacement. Nature 320, 748–750. https://doi.org/10.1038/320748a0.
Green, A.M., Meng, H., Angelaki, D.E., 2007. A reevaluation of the inverse dynamic model for eye movements. J. Neurosci. 27, 1346–1355. https://doi.org/10.1523/jneurosci.3822-06.2007.
Greenstein, D., Lenroot, R., Clausen, L., Chavez, A., Vaituzis, A.C., Tran, L., Gogtay, N., Rapoport, J., 2011. Cerebellar development in childhood onset schizophrenia and non-psychotic siblings. Psychiatry Res. 193, 131–137. https://doi.org/10.1016/j.pscychresns.2011.02.010.
Griffin, J.D., Fletcher, P.C., 2017. Predictive processing, source monitoring, and psychosis. Annu. Rev. Clin. Psychol. 13, 265–289.
Grodd, W., Hülsmann, E., Lotze, M., Wildgruber, D., Erb, M., 2001. Sensorimotor mapping of the human cerebellum: fMRI evidence of somatotopic organization. Hum. Brain Mapp. 13, 55–73. https://doi.org/10.1002/hbm.1025.
Guo, W., Liu, F., Chen, J., Wu, R., Zhang, Z., Yu, M., Xiao, C., Zhao, J., 2015. Resting-state cerebellar-cerebral networks are differently affected in first-episode, drug-naive schizophrenia patients and unaffected siblings. Sci. Rep. 5, 17275. https://doi.org/10.1038/srep17275.
Habas, C., 2012. Functional imaging and the cerebellum: recent developments and challenges. Editorial. Cerebellum 11, 311–313. https://doi.org/10.1007/s12311-012-0375-5.
Habas, C., Kamdar, N., Nguyen, D., Prater, K., Beckmann, C.F., Menon, V., Greicius, M.D., 2009. Distinct cerebellar contributions to intrinsic connectivity networks. J. Neurosci. 29, 8586–8594. https://doi.org/10.1523/JNEUROSCI.1868-09.2009.
Haddock, G., Slade, P.D., Bentall, R.P., 1995. Auditory hallucinations and the verbal transformation effect: the role of suggestions. Pers. Individ. Dif. 19, 301–306. https://doi.org/10.1016/0191-8869(95)00063-C.
Haggard, P., 2017. Sense of agency in the human brain. Nat. Rev. Neurosci. 18, 196–207. https://doi.org/10.1038/nrn.2017.14.
Haggard, P., Miall, R.C., Wade, D., Fowler, S., Richardson, A., Anslow, P., Stein, J., 1995. Damage to cerebellocortical pathways after closed head injury: a behavioural and magnetic resonance imaging study. J. Neurol. Neurosurg. Psychiatry 58, 433–438. https://doi.org/10.1136/jnnp.58.4.433.
Hashimoto, M., Takahara, D., Hirata, Y., Inoue, K.I., Miyachi, S., Nambu, A., Tanji, J., Takada, M., Hoshi, E., 2010. Motor and non-motor projections from the cerebellum to rostrocaudally distinct sectors of the dorsal premotor cortex in macaques. Eur. J. Neurosci. 31, 1402–1413. https://doi.org/10.1111/j.1460-9568.2010.07151.x.
Havermans, R., Honig, A., Vuurman, E.F.P.M., Krabbendam, L., Wilmink, J., Lamers, T., Verheecke, C.J., Jolles, J., Romme, M.A.J., Van Praag, H.M., 1999. A controlled study of temporal lobe structure volumes and P300 responses in schizophrenic patients with persistent auditory hallucinations. Schizophr. Res. 38, 151–158. https://doi.org/10.1016/S0920-9964(99)00006-7.
Heath, R.G., Harper, J.W., 1974. Ascending projections of the cerebellar fastigial nucleus to the hippocampus, amygdala, and other temporal lobe sites. Exp. Neurol. 45, 268–287.
Heath, R.G., Franklin, D.E., Shraberg, D., 1979. Gross pathology of the cerebellum in patients diagnosed and treated as functional psychiatric disorders. J. Nerv. Ment. Dis. 167, 585–592. https://doi.org/10.1097/00005053-197910000-00001.
Heinks-Maldonado, T.H., Mathalon, D.H., Houde, J.F., Gray, M., Faustman, W.O., Ford, J.M., 2007. Relationship of imprecise corollary discharge in schizophrenia to auditory hallucinations. Arch. Gen. Psychiatry 64, 286–296. https://doi.org/10.1001/archpsyc.64.3.286.
Herzfeld, D.J., Kojima, Y., Soetedjo, R., Shadmehr, R., 2018. Encoding of error and learning to correct that error by the Purkinje cells of the cerebellum. Nat. Neurosci. 21, 736–743. https://doi.org/10.1038/s41593-018-0136-y.
Higashima, M., Nagasawa, T., Kawasaki, Y., Oka, T., Sakai, N., Tsukada, T., Koshino, Y., 2003. Auditory P300 amplitude as a state marker for positive symptoms in schizophrenia: cross-sectional and retrospective longitudinal studies. Schizophr. Res. 59, 147–157. https://doi.org/10.1016/S0920-9964(01)00397-8.
Hoffman, R.E., Anderson, A.W., Varanko, M., Gore, J.C., Hampson, M., 2008. Time course of regional brain activation associated with onset of auditory/verbal hallucinations. Br. J. Psychiatry 193, 424–425. https://doi.org/10.1192/bjp.bp.107.040501.
Hoffman, R.E., Fernandez, T., Pittman, B., Hampson, M., 2011. Elevated functional connectivity along a corticostriatal loop and the mechanism of auditory/verbal hallucinations in patients with schizophrenia. Biol. Psychiatry 69, 407–414. https://doi.org/10.1016/j.biopsych.2010.09.050.
Horga, G., Parellada, E., Lomeña, F., Fernández-Egea, E., Mané, A., Font, M., Falcón, C., Konova, A.B., Pavia, J., Ros, D., Bernardo, M., 2011. Differential brain glucose metabolic patterns in antipsychotic-naive first-episode schizophrenia with and without auditory verbal hallucinations. J. Psychiatry Neurosci. 36, 312–321. https://doi.org/10.1503/jpn.100085.
Horga, G., Fernández-Egea, E., Mané, A., Font, M., Schatz, K.C., Falcon, C., Lomeña, F., Bernardo, M., Parellada, E., 2014a. Brain metabolism during hallucination-like auditory stimulation in schizophrenia. PLoS One 9https://doi.org/10.1371/journal.pone.0084987.
Horga, G., Schatz, K.C., Abi-Dargham, A., Peterson, B.S., 2014b. Deficits in predictive coding underlie hallucinations in schizophrenia. J. Neurosci. 34, 8072–8082. https://doi.org/10.1523/JNEUROSCI.0200-14.2014.
Howarth, C., Peppiatt-Wildman, C.M., Attwell, D., 2010. The energy use associated with neural computation in the cerebellum. J. Cereb. Blood Flow Metab. 30, 403–414. https://doi.org/10.1038/jcbfm.2009.231.
Hsu, Y.F., Hämäläinen, J.A., Waszak, F., 2016. The auditory N1 suppression rebounds as prediction persists over time. Neuropsychologia 84, 198–204. https://doi.org/10.1016/j.neuropsychologia.2016.02.019.
Huang, C.M., Liu, G., Huang, R., 1982. Projections from the cochlear nucleus to the cerebellum. Brain Res. 244, 1–8. https://doi.org/10.1016/0006-8993(82)90897-6.
Huang, P., Xi, Y., Lu, Z.L., Chen, Y., Li, X., Li, W., Zhu, X., Cui, L.B., Tan, Q., Liu, W., Li, C., Miao, D., Yin, H., 2015. Decreased bilateral thalamic gray matter volume in first-episode schizophrenia with prominent hallucinatory symptoms: a volumetric MRI study. Sci. Rep. https://doi.org/10.1038/srep14505.
Hughes, G., Desantis, A., Waszak, F., 2013. Attenuation of auditory N1 results from identity-specific action-effect prediction. Eur. J. Neurosci. 37, 1152–1158. https://doi.org/10.1111/ejn.12120.
Hülsmann, E., Erb, M., Grodd, W., 2003. From will to action: sequential cerebellar contributions to voluntary movement. Neuroimage 20, 1485–1492. https://doi.org/10.1016/S1053-8119(03)00307-0.
Hurley, M.J., Mash, D.C., Jenner, P., 2003. Markers for dopaminergic neurotransmission in the cerebellum in normal individuals and patients with Parkinson's disease examined by RT-PCR. Eur. J. Neurosci. 18, 2668–2672. https://doi.org/10.1046/j.

1460-9568.2003.02963.x.
Ichimiya, T., Okubo, Y., Suhara, T., Sudo, Y., 2001. Reduced volume of the cerebellar vermis in neuroleptic-naive schizophrenia. Biol. Psychiatry 49, 20–27. https://doi.org/10.1016/S0006-3223(00)01081-7.
Ide, J.S., Li, C.S., 2011. A cerebellar thalamic cortical circuit for error-related cognitive control. NeuroImage 54, 455–464. https://doi.org/10.1016/j.neuroimage.2010.07.042.
Ikai, Y., Takada, M., Shinonaga, Y., Mizuno, N., 1992. Dopaminergic and non-dopaminergic neurons in the ventral tegmental area of the rat project, respectively, to the cerebellar cortex and deep cerebellar nuclei. Neuroscience 51, 719–728. https://doi.org/10.1016/0306-4522(92)90310-X.
Ikeda, A., Lüders, H.O., Shibasaki, H., Collura, T.F., Burgess, R.C., Morris, H.H., Hamano, T., 1995. Movement-related potentials associated with bilateral simultaneous and unilateral movements recorded from human supplementary motor area. Electroencephalogr. Clin. Neurophysiol. 95, 323–334. https://doi.org/10.1016/0013-4694(95)00086-E.
Ilankovic, L.M., Allen, P.P., Engel, R., Kambeitz, J., Riedel, M., Müller, N., Hennig-Fast, K., 2011. Attentional modulation of external speech attribution in patients with hallucinations and delusions. Neuropsychologia 49, 805–812. https://doi.org/10.1016/j.neuropsychologia.2011.01.016.
Imamizu, H., Kuroda, T., Miyauchi, S., Yoshioka, T., Kawato, M., 2003. Modular organization of internal models of tools in the human cerebellum. Proc. Natl. Acad. Sci. 100, 5461–5466. https://doi.org/10.1073/pnas.0835746100.
Ishikawa, T., Tomatsu, S., Izawa, J., Kakei, S., 2016. The cerebro-cerebellum: could it be loci of forward models? Neurosci. Res. 104, 72–79. https://doi.org/10.1016/j.neures.2015.12.003.
Ito, M., 1970. Neurophysiological aspects of the cerebellar motor control system. Int. J. Neurol. 7, 162–176.
Ito, M., 1984. The modifiable neuronal network of the cerebellum. Jpn. J. Physiol..
Ito, M., 2000. Mechanisms of motor learning in the cerebellum. Brain Res. 886, 237–245. https://doi.org/10.1016/S0006-8993(00)03142-5.
Ito, M., 2006. Cerebellar circuitry as a neuronal machine. Prog. Neurobiol. 78, 272–303. https://doi.org/10.1016/j.pneurobio.2006.02.006.
Ito, M., 2008. Control of mental activities by internal models in the cerebellum. Nat. Rev. Neurosci. 9, 304–313. https://doi.org/10.1038/nrn2332.
Ito, M., 2013. Error detection and representation in the olivo-cerebellar system. Front. Neural Circuits 7, 1–8. https://doi.org/10.3389/fncir.2013.00001.
Ito, M., Yoshida, M., Obata, K., Kawai, N., Udo, M., 1970. Inhibitory control of intracerebellar nuclei by the Purkinje cell axons. Exp. Brain Res. 10, 64–80. https://doi.org/10.1007/BF00340519.
Ivry, R., 2000. Exploring the role of the cerebellum in sensory anticipation and timing: commentary on Tesche and Karhu. Hum. Brain Mapp. 9, 115–118 doi:10.1002/(SICI)1097-0193(200003)9:3 < 115::AID-HBM1 > 3.0.CO;2-5.
Ivry, R.B., Baldo, J.V., 1992. Is the cerebellum involved in learning and cognition? Curr. Opin. Neurobiol. 2, 212–216. https://doi.org/10.1016/0959-4388(92)90015-D.
Ivry, R.B., Keele, S.W., 1989. Timing functions of the cerebellum. J. Cogn. Neurosci. 1, 136–152. https://doi.org/10.1162/jocn.1989.1.2.136.
Jack, B.N., Le Pelley, M.E., Han, N., Harris, A.W.F., Spencer, K.M., Whitford, T.J., 2019. Inner speech is accompanied by a temporally-precise and content-specific corollary discharge. Neuroimage 198, 170–180. https://doi.org/10.1016/j.neuroimage.2019.04.038.
Jardri, R., Pins, D., Bubrovszky, M., Despretz, P., Pruvo, J.P., Steinling, M., Thomas, P., 2007. Self awareness and speech processing: an fMRI study. Neuroimage 35, 1645–1653. https://doi.org/10.1016/j.neuroimage.2007.02.002.
Jardri, R., Pouchet, A., Pins, D., Thomas, P., 2011. Cortical activations during auditory verbal hallucinations in schizophrenia: a coordinate-based meta-analysis. Am. J. Psychiatry 168, 73–81. https://doi.org/10.1176/appi.ajp.2010.09101522.
Johns, L.C., Rossell, S., Frith, C., Ahmad, F., Hemsley, D., Kuipers, E., McGuire, P.K., 2001. Verbal self-monitoring and auditory verbal hallucinations in patients with schizophrenia. Psychol. Med. (Paris) 31, 705–715. https://doi.org/10.1017/S0033291701003774.
Johns, L.C., Allen, P., Valli, I., Winton-Brown, T., Broome, M., Woolley, J., Tabraham, P., Day, F., Howes, O., Wykes, T., McGuire, P., 2010. Impaired verbal self-monitoring in individuals at high risk of psychosis. Psychol. Med. (Paris) 40, 1433–1442. https://doi.org/10.1017/S0033291709991991.
Johnson, J.J., Belyk, M., Schwartze, M., Pinheiro, A.P., Kotz, S.A., 2019. The role of the cerebellum in adaptation: ALE meta-analyses on sensory feedback error. Hum. Brain Mapp. 40, 3966–3981.
Jones, S.R., Fernyhough, C., 2007. Thought as action: inner speech, self-monitoring, and auditory verbal hallucinations. Conscious. Cogn. 16, 391–399. https://doi.org/10.1016/j.concog.2005.12.003.
Kambeitz-Ilankovic, L., Hennig-Fast, K., Benetti, S., Kambeitz, J., Pettersson-Yeo, W., O'daly, O., Mcguire, P., Allen, P., 2013. Attentional modulation of source attribution in first-episode psychosis: a functional magnetic resonance imaging study. Schizophr. Bull. 39, 1027–1036. https://doi.org/10.1093/schbul/sbs101.
Katseff, S., Houde, J., Johnson, K., 2012. Partial compensation for altered auditory feedback: a tradeoff with somatosensory feedback? Lang. Speech 55, 295–308. https://doi.org/10.1177/0023830911417802.
Kawato, M., Gomi, H., 1992a. The cerebellum and VOR/OKR learning models. Trends Neurosci. 15, 445–453. https://doi.org/10.1016/0166-2236(92)90008-V.
Kawato, M., Gomi, H., 1992b. A computational model of four regions of the cerebellum based on feedback-error learning. Biol. Cybern. 68, 95–103. https://doi.org/10.1007/BF00201431.
Kawato, M., Furukawa, K., Suzuki, R., 1987. A hierarchical neural-network model for control and learning of voluntary movement. Biol. Cybern. 57, 169–185. https://doi.org/10.1007/BF00364149.
Kelleher, I., Cannon, M., 2011. Psychotic-like experiences in the general population: characterizing a high-risk group for psychosis. Psychol. Med. (Paris) 41, 1–6. https://doi.org/10.1017/S0033291710001005.
Keller, A., Castellanos, F.X., Vaituzis, A.C., Jeffries, N.O., Giedd, J.N., Rapoport, J.L., 2003. Progressive loss of cerebellar volume in childhood-onset schizophrenia. Am. J. Psychiatry 160, 128–133. https://doi.org/10.1176/appi.ajp.160.1.128.
Kelly, R.M., Strick, P.L., 2003. Cerebellar loops with motor cortex and prefrontal cortex of a nonhuman primate. J. Neurosci. 23, 8432–8444. https://doi.org/10.1523/JNEUROSCI.23-23-08432.2003.
Khilkevich, A., Canton-Josh, J., DeLord, E., Mauk, M.D., 2018. A cerebellar adaptation to uncertain inputs. Sci. Adv. 4https://doi.org/10.1126/sciadv.aap9660. eaap9660.
Kim, D.J., Kent, J.S., Bolbecker, A.R., Sporns, O., Cheng, H., Newman, S.D., Puce, A., O'Donnell, B.F., Hetrick, W.P., 2014. Disrupted modular architecture of cerebellum in schizophrenia: a graph theoretic analysis. Schizophr. Bull. 40, 1216–1226. https://doi.org/10.1093/schbul/sbu059.
Kim, N.Y., Hsu, J., Talmasov, D., Joutsa, J., Soussand, L., Wu, O., Rost, N.S., Morenas-Rodríguez, E., Martí-Fàbregas, J., Pascual-Leone, A., Corlett, P.R., Fox, M.D., 2019. Lesions causing hallucinations localize to one common brain network. Mol. Psychiatry. https://doi.org/10.1038/s41380-019-0565-3.
King, M., Hernandez-Castillo, C.R., Poldrack, R.A., Ivry, R.B., Diedrichsen, J., 2019. Functional boundaries in the human cerebellum revealed by a multi-domain task battery. Nat. Neurosci. 22, 1371–1378.
Kitazawa, S., Kimura, T., Yin, P.B., 1998. Cerebellar complex spikes encode both destinations and errors in arm movements. Nature 392, 494–497. https://doi.org/10.1038/33141.
Knolle, F., Schröger, E., Baess, P., Kotz, S.A., 2012. The cerebellum generates motor-to-auditory predictions: ERP lesion evidence. J. Cogn. Neurosci. 24, 698–706. https://doi.org/10.1162/jocn_a_00167.
Knolle, F., Schröger, E., Kotz, S.A., 2013. Cerebellar contribution to the prediction of self-initiated sounds. Cortex 49, 2449–2461. https://doi.org/10.1016/j.cortex.2012.12.012.
Koreki, A., Maeda, T., Fukushima, H., Umeda, S., Takahata, K., Okimura, T., Funayama, M., Iwashita, S., Mimura, M., Kato, M., 2015. Behavioral evidence of delayed prediction signals during agency attribution in patients with schizophrenia. Psychiatry Res. 230, 78–83. https://doi.org/10.1016/j.psychres.2015.08.023.
Kotz, S.A., Stockert, A., Schwartze, M., 2014. Cerebellum, temporal predictability and the updating of a mental model. Philos. Trans. R. Soc. B Biol. Sci. 369https://doi.org/10.1098/rstb.2013.0403. 20130403–20130403.
Koziol, L.F., Budding, D., Andreasen, N., D'Arrigo, S., Bulgheroni, S., Imamizu, H., Ito, M., Manto, M., Marvel, C., Parker, K., Pezzulo, G., Ramnani, N., Riva, D., Schmahmann, J., Vandervert, L., Yamazaki, T., 2014. Consensus paper: the cerebellum's role in movement and cognition. Cerebellum 13, 151–177. https://doi.org/10.1007/s12311-013-0511-x.
Kühn, S., Nenchev, I., Haggard, P., Brass, M., Gallinat, J., Voss, M., 2011. Whodunnit? Electrophysiological correlates of agency judgements. PLoS One 6. https://doi.org/10.1371/journal.pone.0028657.
Kumari, V., Fannon, D., Ffytche, D.H., Raveendran, V., Antonova, E., Premkumar, P., Cooke, M.A., Anilkumar, A.P.P., Williams, S.C.R., Andrew, C., Johns, L.C., Fu, C.H.Y., McGuire, P.K., Kuipers, E., 2010. Functional MRI of verbal self-monitoring in schizophrenia: performance and illness-specific effects. Schizophr. Bull. 36, 740–755. https://doi.org/10.1093/schbul/sbn148.
Lange, K., 2011. The reduced N1 to self-generated tones: An effect of temporal predictability? Psychophysiology 48, 1088–1095. https://doi.org/10.1111/j.1469-8986.2010.01174.x.
Lange, K., 2013. The ups and downs of temporal orienting: a review of auditory temporal orienting studies and a model associating the heterogeneous findings on the auditory N1 with opposite effects of attention and prediction. Front. Hum. Neurosci. 7, 263. https://doi.org/10.3389/fnhum.2013.00263.
Larøi, F., Sommer, I.E., Blom, J.D., Fernyhough, C., Ffytche, D.H., Hugdahl, K., Johns, L.C., McCarthy-Jones, S., Preti, A., Raballo, A., Slotema, C.W., Stephane, M., Waters, F., 2012. The characteristic features of auditory verbal hallucinations in clinical and nonclinical groups: state-of-the-art overview and future directions. Schizophr. Bull. 38, 724–733. https://doi.org/10.1093/schbul/sbs061.
Lennox, B.R., Park, S.B.G., Medley, I., Morris, P.G., Jones, P.B., 2000. The functional anatomy of auditory hallucinations in schizophrenia. Psychiatry Res. - Neuroimaging 100, 13–20. https://doi.org/10.1016/S0925-4927(00)00068-8.
Leube, D.T., Knoblich, G., Erb, M., Grodd, W., Bartels, M., Kircher, T.T.J., 2003. The neural correlates of perceiving one's own movements. Neuroimage 20, 2084–2090. https://doi.org/10.1016/j.neuroimage.2003.07.033.
Levelt, W.J.M., 1989. Speaking: From Intention to Articulation. MIT Press, Cambridge, MA.
Levelt, W.J.M., 2001. Spoken word production: a theory of lexical access. Proc. Natl. Acad. Sci. U. S. A. 98, 13464–13471. https://doi.org/10.1073/pnas.231459498.
Levitt, J.J., McCarley, R.W., Nestor, P.G., Petrescu, C., Donnino, R., Hirayasu, Y., Kikinis, R., Jolesz, F.A., Shenton, M.E., 1999. Quantitative volumetric MRI study of the cerebellum and vermis in schizophrenia: clinical and cognitive correlates. Am. J. Psychiatry 156, 1105–1107. https://doi.org/10.1176/ajp.156.7.1105.
Liang, K.J., Carlson, E.S., 2019. Resistance, vulnerability and resilience: a review of the cognitive cerebellum in aging and neurodegenerative diseases. Neurobiol. Learn. Mem. https://doi.org/10.1016/j.nlm.2019.01.004.
Linden, D.E.J., Thornton, K., Kuswanto, C.N., Johnston, S.J., Van De Ven, V., Jackson, M.C., 2011. The Brain's voices: comparing nonclinical auditory hallucinations and imagery. Cereb. Cortex 21, 330–337. https://doi.org/10.1093/cercor/bhq097.
Linscott, R.J., van Os, J., 2013. An updated and conservative systematic review and metaanalysis of epidemiological evidence on psychotic experiences in children and adults: on the pathway from proneness to persistence to dimensional expression

across mental disorders. Psychol. Med. (Paris) 43, 1133–1149. https://doi.org/10.1017/S0033291712001626.
Liu, T., Pinheiro, A.P., Deng, G., Nestor, P.G., McCarley, R.W., Niznikiewicz, M.A., 2012. Electrophysiological insights into processing nonverbal emotional vocalizations. Neuroreport 23, 108–112. https://doi.org/10.1097/WNR.0b013e32834ea757.
Loeber, R.T., Cintron, C.M.B., Yurgelun-Todd, D.A., 2001. Morphometry of individual cerebellar lobules in schizophrenia. Am. J. Psychiatry 158, 952–954. https://doi.org/10.1176/appi.ajp.158.6.952.
Lohr, J.B., Jeste, D.V., 1986. Cerebellar pathology in schizophrenia? a neuronometric study. Biol. Psychiatry 21, 865–875. https://doi.org/10.1016/0006-3223(86)90259-3.
Lorenzo, D., Velluti, J.C., Crispino, L., Velluti, R., 1977a. Cerebellar sensory functions: rat auditory evoked potentials. Exp. Neurol. 55, 629–636. https://doi.org/10.1016/0014-4886(77)90289-8.
Lorenzo, D., Velluti, J.C., Crispino, L., Velluti, R., 1977b. Cerebellar sensory functions: rat evoked potentials. Exp. Neurol. 55, 629–636.
Luhrmann, T.M., Alderson-Day, B., Bell, V., Bless, J.J., Corlett, P., Hugdahl, K., Jones, N., Laroi, F., Moseley, P., Padmavati, R., Peters, E., Powers, A.R., Waters, F., 2019. Beyond trauma: a Multiple pathways approach to auditory hallucinations in clinical and nonclinical populations. Schizophr. Bull. 45, S24–S31.
Lupo, M., Troisi, E., Chiricozzi, F.R., Clausi, S., Molinari, M., Leggio, M., 2015. Inability to process negative emotions in cerebellar damage: a functional transcranial doppler sonographic study. Cerebellum 14, 663–669. https://doi.org/10.1007/s12311-015-0662-z.
Mallikarjun, P.K., Lalousis, P.A., Dunne, T.F., Heinze, K., Reniers, R.L., Broome, M.R., Farmah, B., Oyebode, F., Wood, S.J., Upthegrove, R., 2018. Aberrant salience network functional connectivity in auditory verbal hallucinations: a first episode psychosis sample. Transl. Psychiatry 8, 69. https://doi.org/10.1038/s41398-018-0118-6.
Maloku, E., Covelo, I.R., Hanbauer, I., Guidotti, A., Kadriu, B., Hu, Q., Davis, J.M., Costa, E., 2010. Lower number of cerebellar Purkinje neurons in psychosis is associated with reduced reelin expression. Proc. Natl. Acad. Sci. U. S. A. 107, 4407–4411. https://doi.org/10.1073/pnas.0914483107.
Mannarelli, D., Pauletti, C., Currà, A., Marinelli, L., Corrado, A., Delle Chiaie, R., Fattapposta, F., 2019. The Cerebellum Modulates Attention Network Functioning: Evidence from a Cerebellar Transcranial Direct Current Stimulation and Attention Network Test Study. Cerebellum 18, 457–468. https://doi.org/10.1007/s12311-019-01014-8.
Manoliu, A., Riedl, V., Zherdin, A., Mühlau, M., Schwerthöffer, D., Scherr, M., Peters, H., Zimmer, C., Förstl, H., Bäuml, J., Wohlschläger, A.M., Sorg, C., 2014. Aberrant dependence of default mode/central executive network interactions on anterior insular salience network activity in schizophrenia. Schizophr. Bull. 40, 428–437. https://doi.org/10.1093/schbul/sbt037.
Manto, M.U., Jissendi, P., 2012. Cerebellum: links between development, developmental disorders and motor learning Front. Neuroanat. 6, 1. https://doi.org/10.3389/fnana.2012.00001.
Manto, M., Bower, J.M., Conforto, A.B., Delgado-García, J.M., Da Guarda, S.N.F., Gerwig, M., Habas, C., Hagura, N., Ivry, R.B., Marien, P., Molinari, M., Naito, E., Nowak, D.A., Ben Taib, N.O., Pelisson, D., Tesche, C.D., Tilikete, C., Timmann, D., 2012. Consensus paper: roles of the cerebellum in motor control-the diversity of ideas on cerebellar involvement in movement. Cerebellum 11, 457–487. https://doi.org/10.1007/s12311-011-0331-9.
Marcinkiewicz, M., Morcos, R., Chrétien, M., 1989. CNS connections with the median raphe nucleus: Retrograde tracing with WGA-apoHRP-gold complex in the rat. J. Comp. Neurol. 289, 11–35. https://doi.org/10.1002/cne.902890103.
Marvel, C.L., Desmond, J.E., 2010. Functional topography of the cerebellum in verbal working memory. Neuropsychol. Rev. 20, 271–279. https://doi.org/10.1007/s11065-010-9137-7.
Marvel, C.L., Desmond, J.E., 2012. From storage to manipulation: how the neural correlates of verbal working memory reflect varying demands on inner speech. Brain Lang. 120, 42–51. https://doi.org/10.1016/j.bandl.2011.08.005.
Maschke, M., Drepper, J., Kindsvater, K., Kolb, F.P., Diener, H.C., Timmann, D., 2000. Fear conditioned potentiation of the acoustic blink reflex in patients with cerebellar lesions. J. Neurol. Neurosurg. Psychiatry 68, 358–364. https://doi.org/10.1136/jnnp.68.3.358.
McCarthy-Jones, S., Thomas, N., Strauss, C., Dodgson, G., Jones, N., Woods, A., Brewin, C.R., Hayward, M., Stephane, M., Barton, J., Kingdon, D., Sommer, I.E., 2014a. Better than mermaids and stray dogs? Subtyping auditory verbal hallucinations and its implications for research and practice. Schizophr. Bull. 40, S275–S284. https://doi.org/10.1093/schbul/sbu018.
McCarthy-Jones, S., Trauer, T., MacKinnon, A., Sims, E., Thomas, N., Copolov, D.L., 2014b. A new phenomenological survey of auditory hallucinations: evidence for subtypes and implications for theory and practice. Schizophr. Bull. 40, 225–235. https://doi.org/10.1093/schbul/sbs156.
McGuire, P.K., Murray, R.M., Shah, G.M.S., 1993. Increased blood flow in Broca's area during auditory hallucinations in schizophrenia. Lancet 342, 703–706. https://doi.org/10.1016/0140-6736(93)91707-S.
McGuire, P.K., David, A.S., Murray, R.M., Frackowiak, R.S.J., Frith, C.D., Wright, I., Silbersweig, D.A., 1995. Abnormal monitoring of inner speech: a physiological basis for auditory hallucinations. Lancet 346, 596–600. https://doi.org/10.1016/S0140-6736(95)91435-8.
McGuire, P.K., Silbersweig, D.A., Frith, C.D., 1996. Functional neuroanatomy of verbal self-monitoring. Brain 119, 907–917. https://doi.org/10.1093/brain/119.3.907.
McLachlan, N.M., Wilson, S.J., 2017. The contribution of brainstem and cerebellar pathways to auditory recognition. Front. Psychol. 8, 265. https://doi.org/10.3389/fpsyg.2017.00265.
Mechelli, A., Allen, P., Amaro, E., Fu, C.H.Y., Williams, S.C.R., Brammer, M.J., Johns, L.C., McGuire, P.K., 2007. Misattribution of speech and impaired connectivity in patients with auditory verbal hallucinations. Hum. Brain Mapp. 28, 1213–1222. https://doi.org/10.1002/hbm.20341.
Melchitzky, D.S., Lewis, D.A., 2000. Tyrosine hydroxylase- and dopamine transporter-immunoreactive axons in the primate cerebellum: evidence for a lobular- and laminar-specific dopamine innervation. Neuropsychopharmacology 22, 466–472. https://doi.org/10.1016/S0893-133X(99)00139-6.
Miall, R.C., Wolpert, D.M., 1996. Forward models for physiological motor control. Neural Netw. 9, 1265–1279. https://doi.org/10.1016/S0893-6080(96)00035-4.
Miall, R.C., Weir, D.J., Wolpert, D.M., Stein, J.F., 1993. Is the cerebellum a smith predictor? J. Mot. Behav. 25, 203–216. https://doi.org/10.1080/00222895.1993.9942050.
Michie, P.T., 2001. What has MMN revealed about the auditory system in schizophrenia? Int. J. Psychophysiol. 42, 177–194. https://doi.org/10.1016/S0167-8760(01)00166-0.
Middleton, F.A., Strick, P.L., 1994. Anatomical evidence for cerebellar and basal ganglia involvement in higher cognitive function. Science 266 (80-), 458–461. https://doi.org/10.1126/science.7939688.
Middleton, F.A., Strick, P.L., 1997. Dentate output channels: motor and cognitive components. Prog. Brain Res. 114, 553–566. https://doi.org/10.1016/s0079-6123(08)63386-5.
Middleton, F.A., Strick, P.L., 2001. Cerebellar projections to the prefrontal cortex of the primate. J. Neurosci. 21, 700–712.
Mifsud, N.G., Whitford, T.J., 2017. Sensory attenuation of self-initiated sounds maps onto habitual associations between motor action and sound. Neuropsychologia 103, 38–43. https://doi.org/10.1016/j.neuropsychologia.2017.07.019.
Miller, E.K., 2000. The prefontral cortex and cognitive control. Nat. Rev. Neurosci. 1, 59–65. https://doi.org/10.1038/35036228.
Misrahy, G.A., Spradley, J.F., Beran, A.V., Garwood, V.P., 1961. Acoustic cerebellar pathways in cats. J. Neurophysiol. 24, 159–166. https://doi.org/10.1152/jn.1961.24.2.159.
Mitra, J., Snider, R.S., 1969. Cerebellar modification of unitary discharges in auditory system. Exp. Neurol. 23, 341–352. https://doi.org/10.1016/0014-4886(69)90082-X.
Moberget, T., Ivry, R.B., 2019. Prediction, psychosis, and the cerebellum. Biol. Psychiatry Cogn. Neurosci. Neuroimaging 4, 820–831. https://doi.org/10.1016/j.bpsc.2019.06.001.
Moberget, T., Karns, C.M., Deouell, L.Y., Lindgren, M., Knight, R.T., Ivry, R.B., 2008. Detecting violations of sensory expectancies following cerebellar degeneration: a mismatch negativity study. Neuropsychologia 46, 2569–2579. https://doi.org/10.1016/j.neuropsychologia.2008.03.016.
Moberget, T., Alnæs, D., Kaufmann, T., Doan, N.T., Córdova-Palomera, A., Norbom, L.B., Rokicki, J., van der Meer, D., Andreassen, O.A., Westlye, L.T., 2019. Cerebellar gray matter volume is associated with cognitive function and psychopathology in adolescence. Biol. Psychiatry 86, 65–75. https://doi.org/10.1016/j.biopsych.2019.01.019.
Morrison, A.P., Baker, C.A., 2000. Intrusive thoughts and auditory hallucinations: a comparative study of intrusions in psychosis. Behav. Res. Ther. 38, 1097–1106. https://doi.org/10.1016/S0005-7967(99)00143-6.
Morton, S.M., Bastian, A.J., 2006. Cerebellar contributions to locomotor adaptations during splitbelt treadmill walking. J. Neurosci. 26, 9107–9116. https://doi.org/10.1523/JNEUROSCI.2622-06.2006.
Moseley, P., Fernyhough, C., Ellison, A., 2013. Auditory verbal hallucinations as atypical inner speech monitoring, and the potential of neurostimulation as a treatment option. Neurosci. Biobehav. Rev. 37, 2794–2805. https://doi.org/10.1016/j.neubiorev.2013.10.001.
Näätänen, R., Paavilainen, P., Rinne, T., Alho, K., 2007. The mismatch negativity (MMN) in basic research of central auditory processing: A review. Clin. Neurophysiol. 118, 2544–2590. https://doi.org/10.1016/j.clinph.2007.04.026.
Nahab, F.B., Kundu, P., Gallea, C., Kakareka, J., Pursley, R., Pohida, T., Miletta, N., Friedman, J., Hallett, M., 2011. The neural processes underlying self-agency. Cereb. Cortex 21, 48–55. https://doi.org/10.1093/cercor/bhq059.
Nayani, T.H., David, A.S., 1996. The auditory hallucination: a phenomenological survey. Psychol. Med. (Paris) 26, 177–189. https://doi.org/10.1017/S003329170003381X.
Nazimek, J.M., Hunter, M.D., Woodruff, P.W.R., 2012. Auditory hallucinations: expectation-perception model. Med. Hypotheses 78, 802–810. https://doi.org/10.1016/j.mehy.2012.03.014.
Neckelmann, G., Specht, K., Lund, A., Ersland, L., Smievoll, A.I., Neckelmann, D., Hugdahl, K., 2006. MR morphometry analysis of grey matter volume reduction in schizophrenia: association with hallucinations. Int. J. Neurosci. https://doi.org/10.1080/00207450690962244.
Neshige, R., Lüders, H., Friedman, L., Shibasaki, H., 1988. Recording of movement-related potentials from the human cortex. Ann. Neurol. 24, 439–445. https://doi.org/10.1002/ana.410240313.
Nowak, D.A., Hermsdörfer, J., Rost, K., Timmann, D., Topka, H., 2004. Predictive and reactive finger force control during catching in cerebellar degeneration. Cerebellum 3, 227–235. https://doi.org/10.1080/14734220410019057.
Ohmae, S., Medina, J.F., 2015. Climbing fibers encode a temporal-difference prediction error during cerebellar learning in mice. Nat. Neurosci. 18, 1798–1803. https://doi.org/10.1038/nn.4167.
Okugawa, G., Sedvall, G.C., Agartz, I., 2003. Smaller cerebellar vermis but not hemisphere volumes in patients with chronic schizophrenia. Am. J. Psychiatry 160, 1614–1617. https://doi.org/10.1176/appi.ajp.160.9.1614.
Orr, C., Hester, R., 2012. Error-related anterior cingulate cortex activity and the prediction of conscious error awareness. Front. Hum. Neurosci. 6, 177. https://doi.org/10.3389/fnhum.2012.00177.

Panagopoulos, N.T., Papadopoulos, G.C., Matsokis, N.A., 1991. Dopaminergic innervation and binding in the rat cerebellum. Neurosci. Lett. 130, 208–212. https://doi.org/10.1016/0304-3940(91)90398-D.
Pantelis, C., Velakoulis, D., McGorry, P.D., Wood, S.J., Suckling, J., Phillips, L.J., Yung, A.R., Bullmore, E.T., Brewer, W., Soulsby, B., Desmond, P., McGuire, P.K., 2003. Neuroanatomical abnormalities before and after onset of psychosis: a cross-sectional and longitudinal MRI comparison. Lancet 361, 281–288. https://doi.org/10.1016/S0140-6736(03)12323-9.
Parellada, E., Lomena, F., Font, M., Pareto, D., Gutierrez, F., Simo, M., Fernandez-Egea, E., Pavia, J., Ros, D., Bernardo, M., 2008. Fluordeoxyglucose-PET study in first-episode schizophrenic patients during the hallucinatory state, after remission and during linguistic-auditory activation. Nucl. Med. Commun. 29, 894–900. https://doi.org/10.1097/MNM.0b013e328302cd10.
Park, J.-Y., Gu, B.-M., Kang, D.-H., Shin, Y.-W., Choi, C.-H., Lee, J.-M., Kwon, J.S., 2010. Integration of cross-modal emotional information in the human brain: an fMRI study. Cortex 46, 161–169. https://doi.org/10.1016/j.cortex.2008.06.008.
Parvizi, J., 2009. Corticocentric myopia: old bias in new cognitive sciences. Trends Cogn. Sci. (Regul. Ed.) 13, 354–359. https://doi.org/10.1016/j.tics.2009.04.008.
Pastor, M.A., Artieda, J., Arbizu, J., Marti-Climent, J.M., Peñuelas, I., Masdeu, J.C., 2002. Activation of human cerebral and cerebellar cortex by auditory stimulation at 40 Hz. J. Neurosci. 22, 10501–10506. https://doi.org/10.1523/jneurosci.22-23-10501.2002.
Pastor, M.A., Vidaurre, C., Fernández-Seara, M.A., Villanueva, A., Friston, K.J., 2008. Frequency-specific coupling in the cortico-cerebellar auditory system. J. Neurophysiol. 100, 1699–1705. https://doi.org/10.1152/jn.01156.2007.
Paulmann, S., Bleichner, M., Kotz, S.A., 2013. Valence, arousal, and task effects in emotional prosody processing. Front. Psychol. 4, 345. https://doi.org/10.3389/fpsyg.2013.00345.
Pell, M.D., Monetta, L., Paulmann, S., Kotz, S.A., 2009. Recognizing emotions in a foreign language. J. Nonverbal Behav. 33, 107–120. https://doi.org/10.1007/s10919-008-0065-7.
Penhune, V.B., Doyon, J., 2005. Cerebellum and M1 interaction during early learning of timed motor sequences. Neuroimage 26, 801–812. https://doi.org/10.1016/j.neuroimage.2005.02.041.
Perez, V.B., Ford, J.M., Roach, B.J., Loewy, R.L., Stuart, B.K., Vinogradov, S., Mathalon, D.H., 2012. Auditory cortex responsiveness during talking and listening: early illness schizophrenia and patients at clinical high-risk for psychosis. Schizophr. Bull. 38, 1216–1224. https://doi.org/10.1093/schbul/sbr124.
Person, A.L., 2019. Corollary discharge signals in the cerebellum. Biol. Psychiatry Cogn. Neurosci. Neuroimaging 4, 813–819. https://doi.org/10.1016/j.bpsc.2019.04.010.
Petacchi, A., Laird, A.R., Fox, P.T., Bower, J.M., 2005. Cerebellum and auditory function: an ALE meta-analysis of functional neuroimaging studies. Hum. Brain Mapp. 25, 118–128. https://doi.org/10.1002/hbm.20137.
Peters, E.R., Williams, S.L., Cooke, M.A., Kuipers, E., 2012. It's not what you hear, it's the way you think about it: appraisals as determinants of affect and behaviour in voice hearers. Psychol. Med. (Paris) 42, 1507–1514. https://doi.org/10.1017/S0033291711002650.
Pfordresher, P.Q., Mantell, J.T., Brown, S., Zivadinov, R., Cox, J.L., 2014. Brain responses to altered auditory feedback during musical keyboard production: an fMRI study. Brain Res. 1556, 28–37. https://doi.org/10.1016/j.brainres.2014.02.004.
Pinheiro, A.P., Barros, C., Pedrosa, J., 2015. Salience in a social landscape: electrophysiological effects of task-irrelevant and infrequent vocal change. Soc. Cogn. Affect. Neurosci. 11, 127–139. https://doi.org/10.1093/scan/nsv103.
Pinheiro, A.P., Rezaii, N., Rauber, A., Niznikiewicz, M., 2016. Is this my voice or yours? The role of emotion and acoustic quality in self-other voice discrimination in schizophrenia. Cogn. Neuropsychiatry 21, 335–353. https://doi.org/10.1080/13546805.2016.1208611.
Pinheiro, A.P., Rezaii, N., Rauber, A., Nestor, P.G., Spencer, K.M., Niznikiewicz, M., 2017. Emotional self–other voice processing in schizophrenia and its relationship with hallucinations: ERP evidence. Psychophysiology 54, 1252–1265. https://doi.org/10.1111/psyp.12880.
Pinheiro, A.P., Schwartze, M., Kotz, S.A., 2018. Voice-selective prediction alterations in nonclinical voice hearers. Sci. Rep. 8, 14717. https://doi.org/10.1038/s41598-018-32614-9.
Pinheiro, A.P., Farinha-Fernandes, A., Roberto, M.S., Kotz, S.A., 2019. Self-voice perception and its relationship with hallucination predisposition. Cogn. Neuropsychiatry 24, 237–255.
Pinheiro, A.P., Schwartze, M., Amorim, M., Coentre, R., Levy, P., Kotz, S.A., 2020. Changes in motor preparation affect the sensory consequences of voice production in voice hearers. Neuropsychologia.https://doi.org/10.1016/j.neuropsychologia.2020.107531.
Polich, J., 2007. Updating P300: an integrative theory of P3a and P3b. Clin. Neurophysiol. 118, 2128–2148. https://doi.org/10.1016/j.clinph.2007.04.019.
Popa, L.S., Ebner, T.J., 2019. Cerebellum, predictions and errors. Front. Cell. Neurosci. 12, 524. https://doi.org/10.3389/fncel.2018.00524.
Popa, L.S., Streng, M.L., Hewitt, A.L., Ebner, T.J., 2016. The errors of our ways: understanding error representations in cerebellar-dependent motor learning. Cerebellum 15, 93–103. https://doi.org/10.1007/s12311-015-0685-5.
Popa, L.S., Streng, M.L., Ebner, T.J., 2017. Long-term predictive and feedback encoding of motor signals in the simple spike discharge of Purkinje cells. eNeuro 4https://doi.org/10.1523/ENEURO.0036-17.2017.
Powers, A.R., Mathys, C., Corlett, P.R., 2017. Pavlovian conditioning–induced hallucinations result from overweighting of perceptual priors. Science 357 (80-), 596–600. https://doi.org/10.1126/science.aan3458.
Prevosto, V., Sommer, M.A., 2013. Cognitive control of movement via the cerebellar-recipient thalamus. Front. Syst. Neurosci. 7, 56. https://doi.org/10.3389/fnsys.2013.00056.
Raij, T.T., Valkonen-Korhonen, M., Holi, M., Therman, S., Lehtonen, J., Hari, R., 2009. Reality of auditory verbal hallucinations. Brain 132, 2994–3001. https://doi.org/10.1093/brain/awp186.
Ramnani, N., 2006. The primate cortico-cerebellar system: anatomy and function. Nat. Rev. Neurosci. 7, 511–522. https://doi.org/10.1038/nrn1953.
Rapin, L., Dohen, M., Polosan, M., Ferrier, P., Loevenbruck, H., 2013. An EMG study of the lip muscles during covert auditory verbal hallucinations in schizophrenia. J. Speech Lang. Hear. Res. 56, S1882–S1893.
Ratcliffe, M., Wilkinson, S., 2016. How anxiety induces verbal hallucinations. Conscious. Cogn. 39, 48–58. https://doi.org/10.1016/j.concog.2015.11.009.
Repovs, G., Csernansky, J.G., Barch, D.M., 2011. Brain network connectivity in individuals with schizophrenia and their siblings. Biol. Psychiatry 69, 967–973. https://doi.org/10.1016/j.biopsych.2010.11.009.
Requarth, T., Sawtell, N.B., 2014. Plastic corollary discharge predicts sensory consequences of movements in a cerebellum-like circuit. Neuron 82, 896–907. https://doi.org/10.1016/j.neuron.2014.03.025.
Restuccia, D., Marca, G.D., Valeriani, M., Leggio, M.G., Molinari, M., 2007. Cerebellar damage impairs detection of somatosensory input changes. A somatosensory mismatch-negativity study. Brain 130, 276–287. https://doi.org/10.1093/brain/awl236.
Rotarska-Jagiela, A., van de Ven, V., Oertel-Knöchel, V., Uhlhaas, P.J., Vogeley, K., Linden, D.E.J., 2010. Resting-state functional network correlates of psychotic symptoms in schizophrenia. Schizophr. Res. 117, 21–30. https://doi.org/10.1016/j.schres.2010.01.001.
Roth, M.J., Synofzik, M., Lindner, A., 2013. The cerebellum optimizes perceptual predictions about external sensory events. Curr. Biol. https://doi.org/10.1016/j.cub.2013.04.027.
Schlerf, J., Ivry, R.B., Diedrichsen, J., 2012. Encoding of sensory prediction errors in the human cerebellum. J. Neurosci. 32, 4913–4922. https://doi.org/10.1523/JNEUROSCI.4504-11.2012.
Schmahmann, J.D., 1996. From movement to thought: anatomic substrates of the cerebellar contribution to cognitive processing. Hum. Brain Mapp. 4, 174–198 doi:10.1002/(SICI)1097-0193(1996)4:3 < 174::AID-HBM3 > 3.0.CO;2-0.
Schmahmann, J.D., 2004. Disorders of the cerebellum: ataxia, dysmetria of thought, and the cerebellar cognitive affective syndrome J. Neuropsychiatry Clin. Neurosci. 16, 367–378. https://doi.org/10.1176/jnp.16.3.367.
Schmahmann, J.D., 2014. Disorders of the cerebellum: ataxia, dysmetria of thought, and the cerebellar cognitive affective syndrome J. Neuropsychiatry Clin. Neurosci.https://doi.org/10.1176/jnp.16.3.367.
Schmahmann, J.D., 2019. The cerebellum and cognition. Neurosci. Lett. 688, 62–75. https://doi.org/10.1016/j.neulet.2018.07.005.
Schmahmann, J.D., Pandya, D.N., 1991. Projections to the basis pontis from the superior temporal sulcus and superior temporal region in the rhesus monkey. J. Comp. Neurol. 308, 224–248. https://doi.org/10.1002/cne.903080209.
Schmahmann, J.D., Pandya, D.N., 1997. Anatomic organization of the basilar pontine projections from prefrontal cortices in rhesus monkey. J. Neurosci. 17, 438–458.
Schmahmann, J.D., Sherman, J.C., 1998. The cerebellar cognitive affective syndrome. Brain 121, 561–579. https://doi.org/10.1093/brain/121.4.561.
Schmahmann, J.D., Weilburg, J.B., Sherman, J.C., 2007. The neuropsychiatry of the cerebellum - insights from the clinic. Cerebellum 6, 254–267. https://doi.org/10.1080/14734220701490995.
Schmitt, A., Schulenberg, W., Bernstein, H.G., Steiner, J., Schneider-Axmann, T., Yeganeh-Doost, P., Malchow, B., Hasan, A., Gruber, O., Bogerts, B., Falkai, P., 2011. Reduction of gyrification index in the cerebellar vermis in schizophrenia: a post-mortem study. World J. Biol. Psychiatry 12, 99–103. https://doi.org/10.3109/15622975.2010.512090.
Schröger, E., Kotz, S.A., SanMiguel, I., 2015. Bridging prediction and attention in current research on perception and action. Brain Res. 1626, 1–13. https://doi.org/10.1016/j.brainres.2015.08.037.
Schwartze, M., Kotz, S.A., 2013. A dual-pathway neural architecture for specific temporal prediction. Neurosci. Biobehav. Rev. 37, 2587–2596. https://doi.org/10.1016/j.neubiorev.2013.08.005.
Schwartze, M., Tavano, A., Schröger, E., Kotz, S.A., 2012. Temporal aspects of prediction in audition: cortical and subcortical neural mechanisms. Int. J. Psychophysiol. 83, 200–207. https://doi.org/10.1016/j.ijpsycho.2011.11.003.
Schweizer, T.A., Oriet, C., Meiran, N., Alexander, M.P., Cusimano, M., Stuss, D.T., 2007. The cerebellum mediates conflict resolution. J. Cogn. Neurosci. 19, 1974–1982. https://doi.org/10.1162/jocn.2007.19.12.1974.
Scott, M., 2013. Corollary discharge provides the sensory content of inner speech. Psychol. Sci. 24, 1824–1830. https://doi.org/10.1177/0956797613478614.
Scott, M., Yeung, H.H., Gick, B., Werker, J.F., 2013. Inner speech captures the perception of external speech. J. Acoust. Soc. Am. 133https://doi.org/10.1121/1.4794932.EL286-292.
Sellami, L., Bocchetta, M., Masellis, M., Cash, D.M., Dick, K.M., Van Swieten, J., Borroni, B., Galimberti, D., Tartaglia, M.C., Rowe, J.B., Graff, C., Tagliavini, F., Frisoni, G., Finger, E., De Mendonça, A., Sorbi, S., Warren, J.D., Rohrer, J.D., Laforce, R., 2018. Distinct neuroanatomical correlates of neuropsychiatric symptoms in the three main forms of genetic frontotemporal dementia in the GENFI Cohort. J. Alzheimers Dis. 65, 147–163. https://doi.org/10.3233/JAD-180053.
Serino, A., Heydrich, L., Kurian, M., Spinelli, L., Seeck, M., Blanke, O., 2014. Auditory verbal hallucinations of epileptic origin. Epilepsy Behav. 31, 181–186. https://doi.org/10.1016/j.yebeh.2013.12.014.
Shenhav, A., Botvinick, M.M., Cohen, J.D., 2013. The expected value of control: an integrative theory of anterior cingulate cortex function. Neuron 79, 217–240. https://doi.org/10.1016/j.neuron.2013.07.007.
Shergill, S.S., Brammer, M.J., Williams, S.C., Murray, R.M., Mcguire, P.K., 2000. Mapping

auditory hallucinations in schizophrenia using functional magnetic resonance imaging. Arch. Gen. Psychiatry 57, 1033–1038 doi:yoa9342 [pii].
Shergill, S.S., Brammer, M.J., Amaro, E., Williams, S.C.R., Murray, R.M., McGuire, P.K., 2004. Temporal course of auditory hallucinations. Br. J. Psychiatry 185, 516–517. https://doi.org/10.1192/bjp.185.6.516.
Shinn, A.K., Baker, J.T., Cohen, B.M., Öngür, D., 2013. Functional connectivity of left Heschl's gyrus in vulnerability to auditory hallucinations in schizophrenia. Schizophr. Res. 143, 260–268. https://doi.org/10.1016/j.schres.2012.11.037.
Shinn, A.K., Baker, J.T., Lewandowski, K.E., Öngür, D., Cohen, B.M., 2015. Aberrant cerebellar connectivity in motor and association networks in schizophrenia. Front. Hum. Neurosci. 9, 134. https://doi.org/10.3389/fnhum.2015.00134.
Shofer, R.J., Nahvi, M.J., 1969. Firing patterns induced by sound in single units of the cerebellar cortex. Exp. Brain Res. 8, 327–345. https://doi.org/10.1007/BF00234380.
Silbersweig, D.A., Stern, E., Frith, C., Cahill, C., Holmes, A., Grootoonk, S., Seaward, J., McKenna, P., Chua, S.E., Schnorr, L., 1995. A functional neuroanatomy of hallucinations in schizophrenia. Nature 378, 176–179. https://doi.org/10.1038/378176a0.
Simons, C.J.P., Tracy, D.K., Sanghera, K.K., O'Daly, O., Gilleen, J., Dominguez, M., de, G., Krabbendam, L., Shergill, S.S., 2010. Functional magnetic resonance imaging of inner speech in schizophrenia. Biol. Psychiatry 67, 232–237. https://doi.org/10.1016/j.biopsych.2009.09.007.
Smith, Y., Villalba, R., 2008. Striatal and extrastriatal dopamine in the basal ganglia: an overview of its anatomical organization in normal and Parkinsonian brains. Mov. Disord. 23, S534–S547. https://doi.org/10.1002/mds.22027.
Snider, R.S., Maiti, A., 1976. Cerebellar contributions to the papez circuit. J. Neurosci. Res. 2, 133–146. https://doi.org/10.1002/jnr.490020204.
Snider, R.S., Stowell, A., 1944. Receiving areas of tactile, auditory and visual systems in the cerebellum. J. Neurophysiol. 7, 331–357. https://doi.org/10.1152/jn.1944.7.6.331.
Sokolov, A.A., Miall, R.C., Ivry, R.B., 2017. The cerebellum: adaptive prediction for movement and cognition. Trends Cogn. Sci. (Regul. Ed.) 21, 313–332. https://doi.org/10.1016/j.tics.2017.02.005.
Sommer, I.E.C., Diederen, K.M.J., Blom, J.D., Willems, A., Kushan, L., Slotema, K., Boks, M.P.M., Daalman, K., Hoek, H.W., Neggers, S.F.W., Kahn, R.S., 2008. Auditory verbal hallucinations predominantly activate the right inferior frontal area. Brain 131, 3169–3177. https://doi.org/10.1093/brain/awn251.
Sommer, I.E., Daalman, K., Rietkerk, T., Diederen, K.M., Bakker, S., Wijkstra, J., Boks, M.P.M., 2010. Healthy individuals with auditory verbal hallucinations; who are they? Psychiatric assessments of a selected sample of 103 subjects. Schizophr. Bull. 36, 633–641. https://doi.org/10.1093/schbul/sbn130.
Sommer, I.E., Clos, M., Meijering, A.L., Diederen, K.M.J., Eickhoff, S.B., 2012. Resting state functional connectivity in patients with chronic hallucinations. PLoS One 7, e43516. https://doi.org/10.1371/journal.pone.0043516.
Sorg, C., Manoliu, A., Neufang, S., Myers, N., Peters, H., Schwerthöffer, D., Scherr, M., Mühlau, M., Zimmer, C., Drzezga, A., Förstl, H., Bäuml, J., Eichele, T., Wohlschläger, A.M., Riedl, V., 2013. Increased intrinsic brain activity in the striatum reflects symptom dimensions in schizophrenia. Schizophr. Bull. 39, 387–395. https://doi.org/10.1093/schbul/sbr184.
Sorrell, E., Hayward, M., Meddings, S., 2010. Interpersonal processes and hearing voices: A study of the association between relating to voices and distress in clinical and non-clinical hearers. Behav. Cogn. Psychother. 38, 127–140. https://doi.org/10.1017/S1352465809990506.
Stephane, M., 2013. Auditory verbal hallucinations result from combinatoric associations of multiple neural events. Front. Hum. Neurosci. 7, 239. https://doi.org/10.3389/fnhum.2013.00239.
Stone, L.S., Lisberger, S.G., 1986. Detection of tracking errors by visual climbing fiber inputs to monkey cerebellar flocculus during pursuit eye movements. Neurosci. Lett. 72, 163–168. https://doi.org/10.1016/0304-3940(86)90073-X.
Stoodley, C.J., Schmahmann, J.D., 2009. Functional topography in the human cerebellum: a meta-analysis of neuroimaging studies. Neuroimage 44, 489–501. https://doi.org/10.1016/j.neuroimage.2008.08.039.
Stoodley, C.J., Schmahmann, J.D., 2010. Evidence for topographic organization in the cerebellum of motor control versus cognitive and affective processing. Cortex 46, 831–844. https://doi.org/10.1016/j.cortex.2009.11.008.
Streng, M.L., Popa, L.S., Ebner, T.J., 2018. Modulation of sensory prediction error in Purkinje cells during visual feedback manipulations. Nat. Commun. 9, 1099. https://doi.org/10.1038/s41467-018-03541-0.
Sugimori, E., Asai, T., Tanno, Y., 2011. Sense of agency over thought: external misattribution of thought in a memory task and proneness to auditory hallucination. Conscious. Cogn. 20, 688–695. https://doi.org/10.1016/j.concog.2010.12.014.
Sultan, F., Augath, M., Hamodeh, S., Murayama, Y., Oeltermann, A., Rauch, A., Thier, P., 2012. Unravelling cerebellar pathways with high temporal precision targeting motor and extensive sensory and parietal networks. Nat. Commun. 3, 924. https://doi.org/10.1038/ncomms1912.
Swiney, L., Sousa, P., 2014. A new comparator account of auditory verbal hallucinations: how motor prediction can plausibly contribute to the sense of agency for inner speech. Front. Hum. Neurosci. 8, 675. https://doi.org/10.3389/fnhum.2014.00675.
Synofzik, M., Lindner, A., Thier, P., 2008a. The cerebellum updates predictions about the visual consequences of one's behavior. Curr. Biol. 18, 814–818. https://doi.org/10.1016/j.cub.2008.04.071.
Synofzik, Matthis, Vosgerau, G., Newen, A., 2008b. Beyond the comparator model: a multifactorial two-step account of agency. Conscious. Cogn. 17, 219–239. https://doi.org/10.1016/j.concog.2007.03.010.
Synofzik, M., Thier, P., Leube, D.T., Schlotterbeck, P., Lindner, A., 2010. Misattributions of agency in schizophrenia are based on imprecise predictions about the sensory consequences of one's actions. Brain 133, 262–271.
Tachibana, H., Aragane, K., Sugita, M., 1995. Event-related potentials in patients with cerebellar degeneration: electrophysiological evidence for cognitive impairment. Cogn. Brain Res. 2, 173–180. https://doi.org/10.1016/0926-6410(95)90006-3.
Tanaka, H., Ishikawa, T., Lee, J., Kakei, S., 2020. The cerebro-cerebellum as a locus of forward model: a review. Front. Syst. Neurosci. 14, 19. https://doi.org/10.3389/fnsys.2020.00019.
Teramoto, S., Snider, R.S., 1966. Modification of auditory responses by cerebellar stimulation. Exp. Neurol. 16, 191–200. https://doi.org/10.1016/0014-4886(66)90098-7.
Tian, X., Poeppel, D., 2010. Mental imagery of speech and movement implicates the dynamics of internal forward models. Front. Psychol. 1, 166. https://doi.org/10.3389/fpsyg.2010.00166.
Tian, X., Poeppel, D., 2012. Mental imagery of speech: linking motor and perceptual systems through internal simulation and estimation. Front. Hum. Neurosci. 6, 314. https://doi.org/10.3389/fnhum.2012.00314.
Tian, X., Poeppel, D., 2013. The effect of imagination on stimulation: the functional specificity of efference copies in speech processing. J. Cogn. Neurosci. 25, 1020–1036. https://doi.org/10.1162/jocn.
Tian, X., Poeppel, D., 2015. Dynamics of self-monitoring and error detection in speech production: evidence from mental imagery and MEG. J. Cogn. Neurosci. 27, 352–364. https://doi.org/10.1162/jocn_a_00692.
Tian, X., Zarate, J.M., Poeppel, D., 2016. Mental imagery of speech implicates two mechanisms of perceptual reactivation. Cortex 77, 1–12. https://doi.org/10.1016/j.cortex.2016.01.002.
Timm, J., Schönwiesner, M., Schröger, E., SanMiguel, I., 2016. Sensory suppression of brain responses to self-generated sounds is observed with and without the perception of agency. Cortex 80, 5–20. https://doi.org/10.1016/j.cortex.2016.03.018.
Toh, W.L., Thomas, N., Rossell, S.L., 2015. Auditory verbal hallucinations in bipolar disorder (BD) and major depressive disorder (MDD): a systematic review. J. Affect. Disord. 184, 18–28. https://doi.org/10.1016/j.jad.2015.05.040.
Tomatsu, S., Ishikawa, T., Tsunoda, Y., Lee, J., Hoffman, D.S., Kakei, S., 2015. Information processing in the hemisphere of the cerebellar cortex for control of wrist movement. J. Neurophysiol. 115, 255–270. https://doi.org/10.1152/jn.00530.2015.
Tourville, J.A., Reilly, K.J., Guenther, F.H., 2008. Neural mechanisms underlying auditory feedback control of speech. Neuroimage 39, 1429–1443. https://doi.org/10.1016/j.neuroimage.2007.09.054.
Tseng, Y.-w., Diedrichsen, J., Krakauer, J.W., Shadmehr, R., Bastian, A.J., 2007. Sensory prediction errors drive cerebellum-dependent sdaptation of reaching. J. Neurophysiol. 98, 54–62. https://doi.org/10.1152/jn.00266.2007.
Turk, K.W., Flanagan, M.E., Josephson, S., Keene, C.D., Jayadev, S., Bird, T.D., 2018. Psychosis in spinocerebellar ataxias: a case series and study of tyrosine hydroxylase in Substantia Nigra. Cerebellum 17, 143–151. https://doi.org/10.1007/s12311-017-0882-5.
Tzvi, E., Zimmermann, C., Bey, R., Münte, T.F., Nitschke, M., Krämer, U.M., 2017. Cerebellar degeneration affects cortico-cortical connectivity in motor learning networks. Neuroimage Clin. 16, 66–78. https://doi.org/10.1016/j.nicl.2017.07.012.
Van Lutterveld, R., Diederen, K.M.J., Otte, W.M., Sommer, I.E., 2014. Network analysis of auditory hallucinations in nonpsychotic individuals. Hum. Brain Mapp. 35, 1436–1445. https://doi.org/10.1002/hbm.22264.
Van Os, J., Linscott, R.J., Myin-Germeys, I., Delespaul, P., Krabbendam, L., 2009. A systematic review and meta-analysis of the psychosis continuum: evidence for a psychosis proneness-persistence-impairment model of psychotic disorder. Psychol. Med. (Paris) 39, 179–195. https://doi.org/10.1017/S0033291708003814.
Vaughan, S., Fowler, D., 2004. The distress experienced by voice hearers is associated with perceived relationship between the voice hearer and the voice. Br. J. Clin. Psychol. 43, 143–153. https://doi.org/10.1348/014466504323088024.
Vercammen, A., Knegtering, H., den Boer, J.A., Liemburg, E.J., Aleman, A., 2010. Auditory hallucinations in schizophrenia are associated with reduced functional connectivity of the temporo-parietal area. Biol. Psychiatry 67, 912–918. https://doi.org/10.1016/j.biopsych.2009.11.017.
Verdoux, H., Van Os, J., 2002. Psychotic symptoms in non-clinical populations and the continuum of psychosis. Schizophr. Res. 54, 59–65. https://doi.org/10.1016/S0920-9964(01)00352-8.
Wang, X.F., Woody, C.D., Chizhevsky, V., Gruen, E., Landeira-Fernandez, J., 1991. The dentate nucleus is a short-latency relay of a primary auditory transmission pathway. Neuroreport 2, 361–364. https://doi.org/10.1097/00001756-199107000-00001.
Waters, F., Fernyhough, C., 2017. Hallucinations: a systematic review of points of similarity and difference across diagnostic classes. Schizophr. Bull. 43, 32–43. https://doi.org/10.1093/schbul/sbw132.
Waters, F., Jablensky, A., 2009. Time discrimination deficits in schizophrenia patients with first-rank (passivity) symptoms. Psychiatry Res. 167, 12–20. https://doi.org/10.1016/j.psychres.2008.04.004.
Waters, F.A.V., Badcock, J.C., Michie, P.T., Maybery, M.T., 2006. Auditory hallucinations in schizophrenia: intrusive thoughts and forgotten memories. Cogn. Neuropsychiatry 11, 65–83. https://doi.org/10.1080/13546800444000191.
Whitford, T.J., Mathalon, D.H., Shenton, M.E., Roach, B.J., Bammer, R., Adcock, Ra, Bouix, S., Kubicki, M., De Siebenthal, J., Rausch, a C., Schneiderman, J.S., Ford, J.M., 2011. Electrophysiological and diffusion tensor imaging evidence of delayed corollary discharges in patients with schizophrenia. Psychol. Med. (Paris) 41, 959–969. https://doi.org/10.1017/S0033291710001376.
Whitford, T.J., Jack, B.N., Pearson, D., Griffiths, O., Luque, D., Harris, A.W., Spencer, K.M., Le Pelley, M.E., 2017. Neurophysiological evidence of efference copies to inner speech. Elife 6, e28197. https://doi.org/10.7554/eLife.28197.
Wilkinson, S., Fernyhough, C., 2017. Auditory verbal hallucinations and inner speech: a predictive processing perspective. In: Radman, Z. (Ed.), Before Consciousness: In Search of the Fundamentals of Mind. Imprint Academics, Exeter, UK. https://doi.org/10.26530/oapen_1001597.

Winton-Brown, T.T., Fusar-Poli, P., Ungless, M.A., Howes, O.D., 2014. Dopaminergic basis of salience dysregulation in psychosis. Trends Neurosci. 37, 85–94. https://doi.org/10.1016/j.tins.2013.11.003.

Wiser, A.K., Andreasen, N.C., O'Leary, D.S., Watkins, G.L., Boles Ponto, L.L., Hichwa, R.D., 1998. Dysfunctional cortico-cerebellar circuits cause "cognitive dysmetria" in schizophrenia. Neuroreport. https://doi.org/10.1097/00001756-199806010-00042.

Witter, L., De Zeeuw, C.I., 2015. Regional functionality of the cerebellum. Curr. Opin. Neurobiol. 33, 150–155. https://doi.org/10.1016/j.conb.2015.03.017.

Wolf, N.D., Sambataro, F., Vasic, N., Frasch, K., Schmid, M., Schönfeldt-Lecuona, C., Thomann, P.A., Wolf, R.C., 2011. Dysconnectivity of multiple resting-state networks in patients with schizophrenia who have persistent auditory verbal hallucinations. J. Psychiatry Neurosci. 36, 366–374. https://doi.org/10.1503/jpn.110008.

Wolfe, J.W., 1972. Responses of the cerebellar auditory area to pure tone stimuli. Exp. Neurol. 36, 295–309. https://doi.org/10.1016/0014-4886(72)90025-8.

Wolpert, D.M., Ghahramani, Z., 2000. Computational principles of movement neuroscience. Nat. Neurosci. 3, 1212–1217. https://doi.org/10.1038/81497.

Wolpert, D.M., Miall, R.C., Kawato, M., 1998. Internal models in the cerebellum. Trends Cogn. Sci. (Regul. Ed.) 2, 338–347. https://doi.org/10.1016/S1364-6613(98)01221-2.

Wu, T., Wang, L., Hallett, M., Chen, Y., Li, K., Chan, P., 2011. Effective connectivity of brain networks during self-initiated movement in Parkinson's disease. Neuroimage 55, 204–215. https://doi.org/10.1016/j.neuroimage.2010.11.074.

Xu-Wilson, M., Chen-Harris, H., Zee, D.S., Shadmehr, R., 2009. Cerebellar contributions to adaptive control of Saccades in humans. J. Neurosci. 29, 12930–12939. https://doi.org/10.1523/JNEUROSCI.3115-09.2009.

Yamaguchi, S., Tsuchiya, H., Kobayashi, S., 1998. Visuospatial attention shift and motor responses in cerebellar disorders. J. Cogn. Neurosci. 10, 95–107. https://doi.org/10.1162/089892998563806.

Yao, B., Belin, P., Scheepers, C., 2011. Silent reading of direct versus indirect speech activates voice-selective areas in the auditory cortex. J. Cogn. Neurosci. 23, 3146–3152. https://doi.org/10.1162/jocn_a_00022.

Yao, B., Belin, P., Scheepers, C., 2012. Brain "talks over" boring quotes: top-down activation of voice-selective areas while listening to monotonous direct speech quotations. Neuroimage 60, 1832–1842. https://doi.org/10.1016/j.neuroimage.2012.01.111.

Zhang, S., Sun, X., Jen, P.H.S., 1990. HRP study of neural projections of acoustic signal to the cerebellum of echolocating bats. Acta Theriol. Sin. 10, 110–113.

Zhao, Z., Li, X., Feng, G., Shen, Z., Li, S., Xu, Y., Huang, M., Xu, D., 2018. Altered effective connectivity in the default network of the brains of first-episode, drug-naïve schizophrenia patients with auditory verbal hallucinations. Front. Hum. Neurosci. 12, 456.

Zheng, Z.Z., Vicente-Grabovetsky, A., MacDonald, E.N., Munhall, K.G., Cusack, R., Johnsrude, I.S., 2013. Multivoxel patterns reveal functionally differentiated networks underlying auditory feedback processing of speech. J. Neurosci. 33, 4339–4348. https://doi.org/10.1523/JNEUROSCI.6319-11.2013.

Zhu, L., Scelfo, B., Hartell, N.A., Strata, P., Sacchetti, B., 2007. The effects of fear conditioning on cerebellar LTP and LTD. Eur. J. Neurosci. 26, 219–227. https://doi.org/10.1111/j.1460-9568.2007.05632.x.

Zhu, L., Sacco, T., Strata, P., Sacchetti, B., 2011. Basolateral amygdala inactivation impairs learning-induced long-term potentiation in the cerebellar cortex. PLoS One 6, e16673. https://doi.org/10.1371/journal.pone.0016673.